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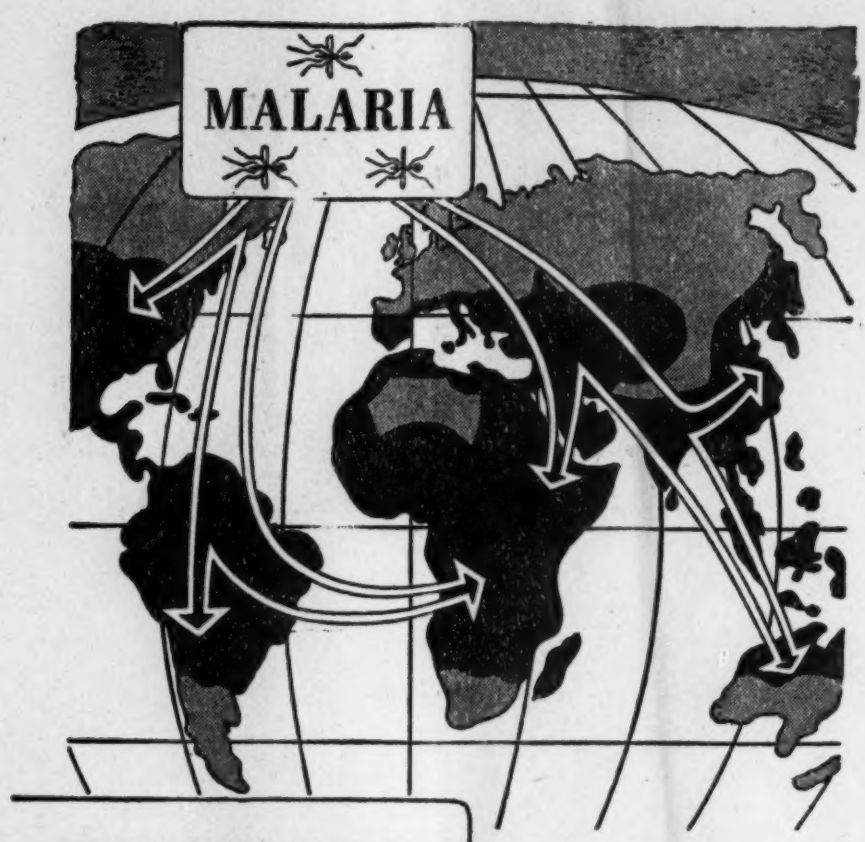

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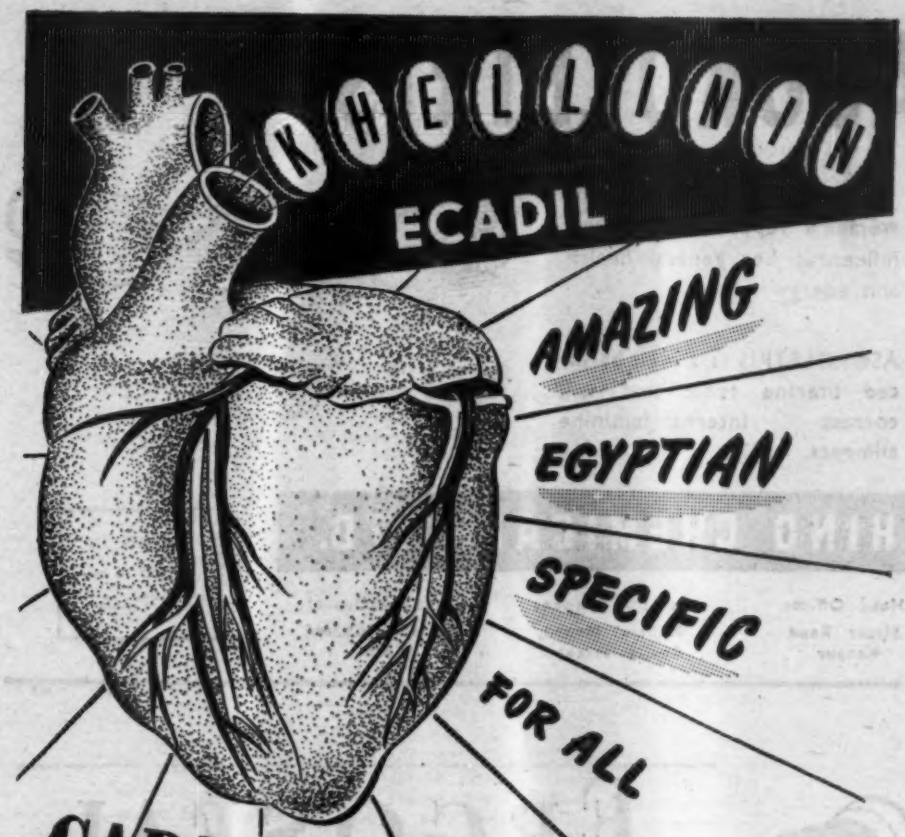

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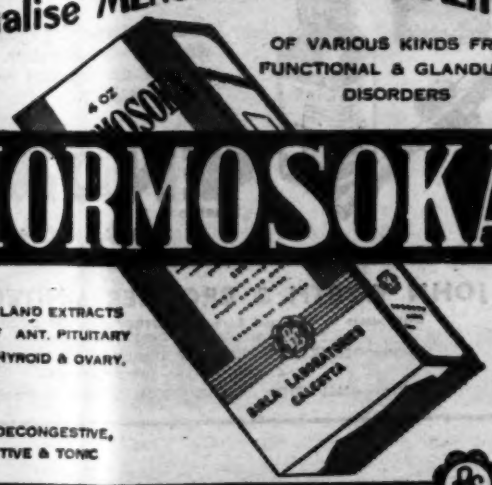

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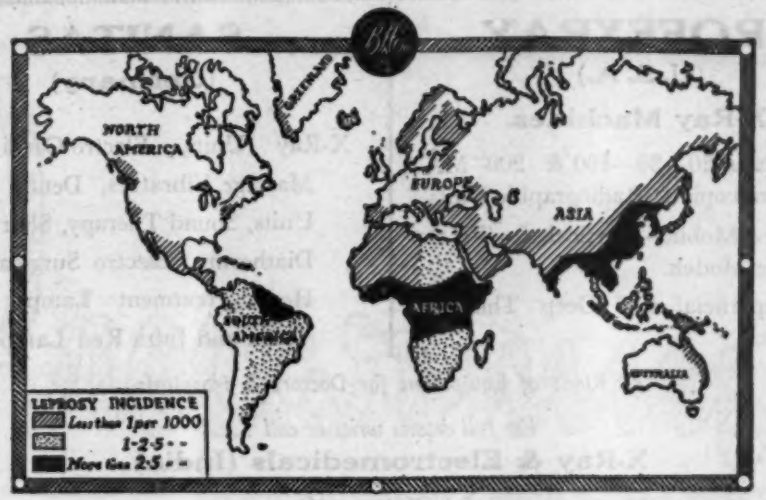

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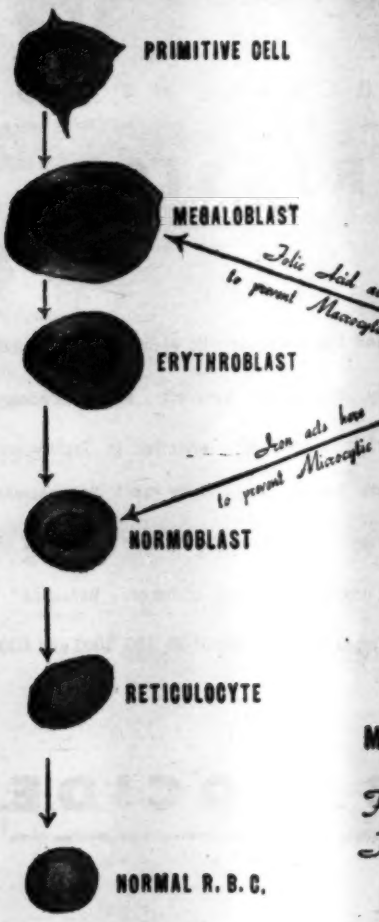

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STUDIES ON ŒDEMA IN TOXÆMIA OF PREGNANCY*

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Oedema has long been recognised as an important manifestation of toxæmia of pregnancy. Recent studies on weight changes during gestation have shown that occult œdema frequently precedes hypertension by several weeks. The capacity of the interstitial tissue to hold water is enormous. In normal individuals this interstitial water constitutes about 15 per cent of the body weight.

The importance of abnormal gain in weight in pregnancy toxæmia was first recognised by Zangemeister (1903). A considerable volume of literature has accumulated on this subject in recent years and although all authors are not in agreement, most investigators (Bingham, 1932; Harding and Van Wyck, 1932; Cummings, 1937; Evans, 1937) believe that an abnormal increase of the volume of the interstitial water often precedes the clinical manifestations of the toxæmia. Later investigations conducted with the estimation of the thiocyanate available water, especially those of Chesley and Chesley (1943) leave little doubt about this contention. The object of the present investigation is to determine what causes this abnormal retention of water in the tissue spaces. In a previous communication the writer (Mukherjee and Govan, 1950) has explained the biochemical mechanism and electrolytic disturbance involved in this water-shift from the plasma into the tissue spaces. The results of an inquiry into some of the relevent clinical conditions are presented here.

1. Hypertension and Oedema

It is generally agreed that hypertension is the earliest clinical manifestation of toxæmia of pregnancy, although a regular and periodic check on the body-weight demonstrates that an abnormal weight-gain sometimes precedes hypertension by several weeks. Browne (1944) points out that hypertension and œdema are frequently complementary in nature in deciding the onset of eclamptic convulsions.

The only reference available in the literature which shows the relation between hypertension and œdema in pre-eclampsia is the report by Chesley and Chesley (*loc. cit.*). These authors observed that in the presence of a diastolic pressure of 90 mm. Hg. or more, an abnormal increase of the interstitial water caused severe toxæmia in 72 per cent of cases. Although this observation may be clinically helpful, the authors made no attempt to determine if a rise of the blood pressure *per se* in toxæmia can give rise to œdema by increasing the filtration pressure, or by interfering with the permeability of the capillaries. From a study of hypertension in general this seems unlikely, but since the hypertension of pregnancy toxæmia differs in some important respects from that seen in general medical practice the possibility of any existing relationship should be considered.

Table 1—Showing the Relation of Oedema to Blood Pressure in Pre-Eclampsia

Degree of œdema	No. of cases	Systolic Pressure mm. Hg.	Diastolic Pressure mm. Hg.
±	8	149·6±0·87 (148—150)	96.7±0·75· (90—100)
1+	29	150·0±10·6 (140—180)	99·1±6·5 (90—110)
2+	49	160·1±14·5 (145—210)	104·1±7·2 (90—120)
3+	19	168·8±19·0 (140—210)	105·3±6·7 (95—126)

For this purpose, data obtained from 100 cases of pre-eclampsia have been analysed. Table 1 shows the blood pressure values obtained in different degrees of œdema. In all cases the readings were taken by the writer himself just after 24 hours of complete rest in bed. The figures generally show that with increasing blood pressure the œdema tends to increase. Careful analysis, however, reveals that the variability of the blood pressure at each stage of œdema is marked, and that there is no constant relationship between the height of blood pressure and the degree of œdema. This is also borne out by Table 2, where the pro-

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TABLE 2—SHOWING THE RELATION OF SYSTOLIC AND DIASTOLIC BLOOD PRESSURE TO OEDEMA

Systolic B.P.	*Degree of Œdema* 1+	2+	3+	*Diastolic B.P.*	*Degree of Œdema* 1+	2+	3+
140—195 ..	21 (72·4%)	24 (49·3%)	5 (26·3%)	Less than 90 ..	4 (13·8%)	1 (2·0%)	—
160—179 ..	7 (24·1%)	21 (42·7%)	8 (42·2%)	90—100 ..	21 (72·4%)	26 (53·1%)	9 (47·4%)
180—199 ..	1 (3·5%)	3 (6·1%)	5 (26·3%)	101—110 ..	4 (13·8%)	16 (32·7%)	6 (31·6%)
200+ ..	—	1 (2·0%)	1 (5·3%)	111—120 ..	—	6 (12·2%)	4 (21·0%)

cedure of analysis has been reversed, and the degree of œdema has been correlated to the levels of blood pressure. The table shows that, although there is a greater percentage of cases with marked œdema at the higher ranges of blood pressure, cases with only slight hypertension frequently show gross œdema. For instance, in 26·3 per cent of cases with anasarca the systolic blood pressure was less than 160 mm. Hg. Nevertheless, when the systolic pressure was 180 mm., or more, for each case of slight (1+) œdema there were 2 cases of moderate (2+), and 9 cases of severe (3+) œdema. When the diastolic pressure was more than 110 mm. Hg. for every 2 cases of moderate œdema, there were 3 cases of severe œdema, while no case of slight œdema was seen in this group (Fig. 1.).

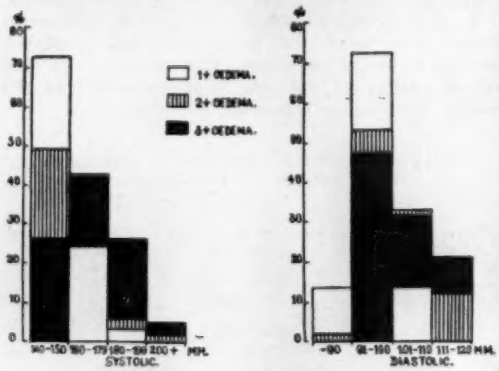

FIG. 1—The incidence of various degrees of œdema in relation to different levels of systolic and diastolic blood pressures.

Theoretically, uncomplicated high blood pressure would increase the filtration force at the proximal end of the capillary bed, but under such circumstances reabsorption of fluid from the tissue spaces is also increased, so that a balance between filtration and reabsorption is established at a higher level without causing any increase of the interstitial water. If hypertension, *per se*, were to cause œdema in toxæmia of pregnancy, then this mechanism must be accompanied by a hindrance to the process of reabsorption, which is governed by the concentration of the proteins in the plasma, and that of the electrolytes on either side of the capillary membrane. That hypertension itself plays only a minor role in the ætiology of the toxæmic œdema of pregnancy is shown by the fact that, in these patients during early puerperium the œdema rapidly disappears, while blood pressure still persists at a high level. Studies on the thiocyanate available water have also failed to show any relationship between the increase in the interstitial tissue water and the level of hypertension in toxæmia of pregnancy.

2. ALBUMINURIA AND OEDEMA

Albuminuria is a frequent finding in toxæmia of pregnancy. Dieckmann (1941) states that noticeable albuminuria was found on more than 2 occasions in 59 per cent of mild, and 94 per cent of severe pre-eclamptic patients. Albuminuria is also an important feature of nephritis complicating pregnancy (92 per cent, Dieckmann, 1941). Therefore in a study of a relation between albuminuria and œdema, cases of nephritis require careful elimination. For the purpose of the present investigation 100 cases of pre-eclampsia of varying severity where the urea clearance was not less than 90 per cent of normal, were selected. In all cases Addis' count and urea concentration test were also done, and any patient who presented a doubtful or abnormal finding was discarded. The results obtained are shown in Table 3.

TABLE 3—SHOWING THE RELATION OF OEDEMA TO ALBUMINURIA

Degree of Œdema	*Albuminuria: Gm. per litre.* Tr.—2	2—4	4—6	6—8	8—10	10+
± ..	3(16·6%)	—	—	—	—	—
1+ ..	12(66·7%)	12(36·4%)	4(14·3%)	—	—	—
2+ ..	3(16·6%)	20(60·6%)	18(64·5%)	3(42·8%)	3(33·3%)	1(25·0%)
3+ ..	—	1(3·0%)	6(21·4%)	4(57·2%)	6(66·7%)	4(75·0%)

Albuminuria appears to have a more direct relationship to œdema than hypertension. With less than 0·2 per cent albumin in the urine more than 80 per cent of the patients showed either a trace or slight œdema, and in none was the œdema of a generalised nature. On the other hand, when there was 0·6 per cent or more of albumin in the urine, the œdema was in all cases of moderate or marked severity. The figures in the table clearly depict a shift to the right of the degree of albuminuria as the œdema increases in severity. The obvious conclusion presented by these figures however, require to be considered in the light of the findings in two types of cases.

1. Normal pregnant women, who are occasionally seen to develop œdema of the inferior extremities, and less frequently of the abdomen. As a rule they have neither hypertension nor albuminuria.

2. Oedema of true pre-eclamptic patients which rapidly subsides after confinement. Kobes (1930) described a condition called delayed healing, in which the albuminuria persists for a long time after the pregnancy is over and this condition is seldom, if ever associated with œdema.

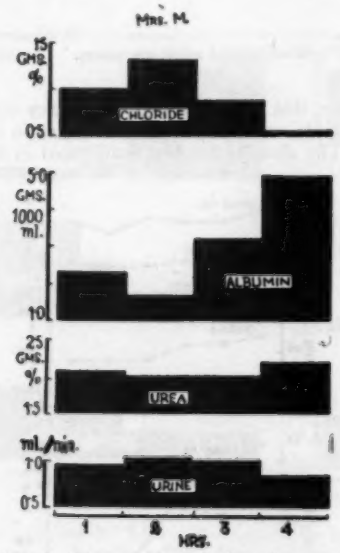

FIG. 2—Diagram of the findings from an illustrative case showing that in a case of pre-eclampsia a fall in the urinary output is attended with an increase of albuminuria. These changes are associated with a diminution of the excretion of chlorides in the urine, but the urea concentrating power of the kidneys is not grossly affected.

Thus, in spite of a marked general relationship between albuminuria and œdema it is difficult to believe that albuminuria could be a direct cause of œdema in pregnancy toxæmia. However, in this connection two important facts deserve consideration.

As early as 1812, Wells demonstrated that proteinuria varies directly with diuresis. This has been confirmed by numerous subsequent investigator (Berglund, Scriver and Medes, 1935). It is commonly found that the subsidence of œdema is accompanied by marked diuresis and a rapid disappearance of albuminuria. Conversely, an increase of œdema is associated with oliguria, during which stage, albuminuria markedly increases (Fig. 2). The apparent relationship which has been observed to exist between albuminuria and œdema probably depicts the relation between oliguria and œdema, the albuminuria assuming an importance because of its association with decreased urinary output. Facts in support of this belief will be presented in the following section.

The source of the protein in the urine is also an important factor in the evaluation of any relationship between albuminuria and œdema. It is generally believed that in presence of albuminuria the protein is derived from the plasma. Recent studies on electrophoresis of urinary and plasma proteins by Luetscher (1940) corroborate this. Continual loss of protein in the urine causes hypoproteinæmia, and thereby a reduction of the oncotic pressure of the plasma. Strauss (1938) and other workers are of the opinion that this reduction of the colloid osmotic pressure of the plasma is the immediate cause of œdema. This will form the subject of discussion in a subsequent section. However, it can be stated now that the existing evidences are too slender to believe that albuminuria *per se* could be the cause of the œdema in pre-eclamptic toxæmia.

3. Oliguria and Oedema

Conditions associated with œdema are also accompanied with oliguria. Congestive cardiac failure, acute nephritis, nephrotic syndrome and severe anæmias provide examples in point. But the contrary does not always follow. All cases of oliguria and anuria do not show œdema. Calculus anuria, anuria following ligature of the ureters, experimental or surgical ablation of the kidneys usually cause either complete, or nearly complete anuria without any recognisable œdema. Bilateral renal cortical necrosis also causes marked oliguria, and even anuria, but œdema is never a prominent feature in this condition.

In toxæmia of pregnancy oliguria is one of the earliest symptoms. However, a study of this is difficult, because the patient does not often notice the symptom at its earliest stages. In an attempt to study the relationship between oliguria and œdema 20 cases of pre-eclampsia were closely studied. In the selection of these cases precaution was observed in order not to include any case of nephritis or essential hypertension. In every patient the investigation covered a period of 6 to 7 days, during which the œdema improved in 8 cases, appeared to remain stationary in 6, and appreciably increased in 6 patients. The degree of œdema was judged by measuring the girth of the ankle at the intermalleolar region. Twenty-four hour specimens of urine were collected, and the output expressed as ml. of urine secreted per minute each day.

At the same time the packed-cell-volume of the blood was recorded. Table 4 shows the results obtained in this investigation (average ± probable error, minimum and maximum values).

TABLE 4—SHOWING THE RELATION OF OEDEMA TO HAEMOCONCENTRATION AND OLIGURIA

Days	IMPROVED			DETERIORATED			STATIONARY		
	Ankle girth cm.	Urine ml. min	Hæmato-crit %	Ankle girth cm.	Urine ml. min.	Hæmato-crit %	Ankle girth cm.	Urine ml. min.	Hæmato-crit %
1	29·6±2·8 (34·7—25·0)	0·508±0·079 (0·605—0·848)	39·8±1·8 (41·8—35·5)	25·0±2·8 (30·4—22·0)	1·008±0·11 (1·105—0·666)	35·7±1·7 (38·5—31·4)	27·1±3·2 (35·5—22·8)	0·962±0·095 (1·124—0·781)	36·4±1·5 (38·0—34·6)
2	28·5±2·0 (33·0—24·8)	0·505±0·067 (0.600—0·860)	38·9±1·5 (41·8—35·4)	24·9±2·5 (30·0—22·0)	0·987±0·12 (1·107—0·660)	35·9±1·8 (38·8—32·0)	26·4±2·8 (34·0—22·4)	0.986±0·067 (1·110—0·758)	36·4±1·8 (37·8—34·9)
3	25·9±1·5 (30·0—22·1)	0·525±0·067 (0·621—0·886)	35·5±2·5 (40·0—31·0)	25·3±2·8 (30·9—22·6)	0·881±0·37 (1·100—0·514)	36·6±1·1 (39·2—34·6)	26·0±2·6 (31·5—22·6)	0·981±0·087 (1·058—0·760)	36·6±1·4 (38·0—35·0)
4	28·6±2·8 (27·4—21·0)	0·914±0·193 (1·105—0·567)	31·6±1·5 (34·5—28·0)	26·0±2·6 (31·2—23·4)	0·749±0·12 (0·910—0·415)	37·7±1·1 (39·6—35·8)	25·4±1·7 (29·5—22·5)	0·948±0·098 (1·128—0·775)	36·2±1·6 (38·0—34·1)
5	22·1±1·1 (27·4—21·0)	1·197±0·110 (1·350—1·070)	30·9±0·76 (33·4—29·0)	26·6±2·1 (31·8—24·4)	0·635±0·14 (0·884—0·410)	38·8±0·85 (40·4—37·1)	25·1±1·9 (28·0—22·5)	0·907±0·088 (1·108—0·780)	36·8±1·8 (37·8—35·0)
6	21·5±0·95 (25·0—20·0)	1·479±0·110 (1·800—1·200)	31·8±0·85 (32·6—30·7)	27·5±2·6 (33·8—24·0)	0·548±0·097 (0·750—0·398)	40·4±0·80 (41·9—39·3)	25·1±1·7 (28·5—22·5)	0·940±0·058 (1·100—0·850)	36·7±1·2 (37·9—34·8)
7	21·3±0·67 (22·0—19·6)	1·629±0·120 (1·818—1·430)	32·4±0·32 (33·6—32·0)	—	—	—	—	—	—

Figures supplied in this table are average ± probable error : maximum and minimum values.

The interesting feature of this study is that the urinary output and the degree of œdema followed each other closely in an inverse ratio. This, however, does not indicate that the oliguria in pregnancy toxæmia is the immediate cause of retention of water in the tissue spaces. The changes are best manifested in the group

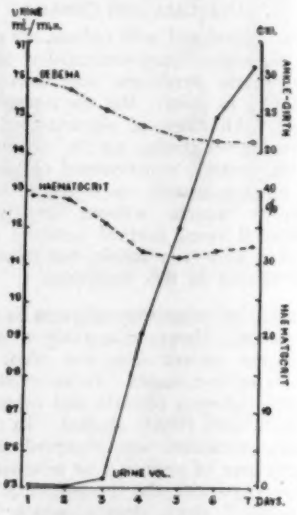

FIG. 3—Diagram showing the correlation between the urinary output and hæmoconcentration and the degree of œdema present in a case of pre-eclampsia. The figure shows the behaviour when the œdema gradually improves.

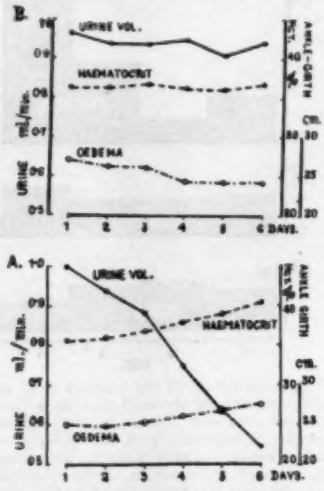

FIG. 4—The correlation between the urinary output and hæmoconcentration and pre-eclamptic œdema.
A. Findings from cases where the œdema increased during the period of investigation.
B. Similar findings from cases where the œdema remained stationary.

where œdema subsided. By the third day the girth of the ankle had diminished by 2·6 cm., while the change in the urinary output was only slight. On the following day the ankle girth was less by a further 2·3 cm., and the diuresis was only just beginning. In other words, marked improvement of the œdema occurred before the urinary output showed any striking change. During the subsequent period the gradient of the urinary output was more marked than that of the associated change in the œdema (Fig. 3). This relationship between œdema and oliguria which is reversely manifested in the other two groups as well (Fig. 4), indicates that oliguria is not the primary cause of œdema, and a consequent diversion of fluid into the interstitial tissues of the body. Further evidence in support of this view is found in the hæmatocrit readings. The improved cases in Table 4 show that the hæmatocrit fell by 3·4 per cent before diuresis commenced, and the maximum decrease of the packed-cell volume occurred an appreciable time before the maximum diuresis occurred. A similar (although a less noticeable, because of the nature of these cases) but inverse change was also noticed in the other two groups of cases (Fig. 5). These findings clearly suggest

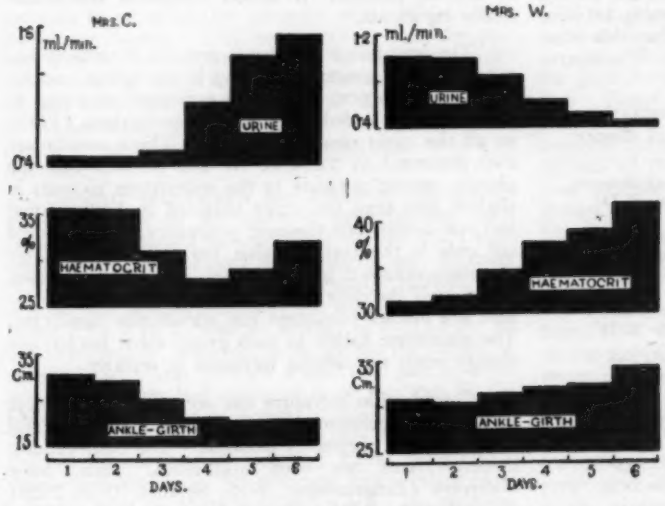

FIG. 5—A and B. Typical illustrative cases showing the mutual relationship between œdema, hæmoconcentration and urinary output in pre-eclamptic toxæmia.

that oliguria follows, and does not precede the diversion of fluid from the plasma into the tissue spaces. This exposes the possibility that the oliguria of pregnancy toxæmia is not the cause, but partly an indirect effect of the œdema. The latter gives rise to hæmoconcentration, and calls for compensatory mechanisms directed toward the conservation of the water content of the plasma. A mechanism such as this would explain the alterations in hæmodynamics affecting the kidneys, the details of which will be presented in a subsequent communication.

4. Plasma Volume and Oedema

Plasma volume has been estimated by several investigators during pregnancy. The values presented by different workers show only a small range of variation. Dieckmann and Wegner (1934) found the average plasma volume in normal pregnancy to be 47 ml. per kilogram of the body weight. From the data provided by Roscoe and Donaldson (1946) the average appears to be 48 ml. The present investigation is based on 26 cases of normal pregnancy between 36 weeks and term, and 45 cases of pre-eclampsia with varying degrees of œdema. Plasma volume was estimated by the injection of Evan's blue (T 1824), and the method employed was that of Davies (1942).

The average plasma volume in the normal cases was 49·45 ml. (standard deviation 3·22), per kilogram of the body weight. The maximum and the minimum values in this series were 52·8 and 44·7 ml. respectively. The results are shown in Table 5.

Oedema was associated with a decrease in the plasma volume not only in the average, but also in the minimum and maximum in each group. The decline in the volume of the circulating plasma is progressive as œdema increases in severity. The average for all cases of pre-eclamptic œdema was 42·6 ml. (standard deviation 2·8) per kilogram of the body weight. It will be seen that the difference between the normal and pre-eclamptic values is statistically significant.

The average value for the plasma volume in pre-eclampsia was found by Dieckmann (1941) to be 39 ml. per kilogram, and this compares favourably with the value obtained in the present series. The significance of the change in the plasma volume is realised when this is compared with the change in the hæmatocrit readings described in the last section. The fall in the plasma volume, together with an increase in the hæmatocrit value while the œdema increases in severity, indicates that the œdema in pregnancy toxæmia is caused by a diversion of the plasma water from the intra-vascular into the extra-vascular tissue spaces. The relation of oliguria to œdema is best studied in this perspective. Had oliguria been a primary cause of œdema it would have been natural to expect a phase of hæmodilution before œdema was manifest. The fact that the plasma volume falls and the hæmatocrit rises inspite of oliguria provides the conclusion that the œdema is not a passive process caused by decreased elimination of fluid by the kidneys. Clinical evidences also corroborate this view, for renal diuretics never succeed in increasing the urinary output, and/or decreasing the œdema in pre-eclamptic toxæmia.

5. Plasma Proteins and Oedema

The immediate physical effect of the loss of water

from the plasma into the tissues is the opposite of that caused by the loss of protein from the plasma into the urine. This should counteract the process which causes fluid to migrate into the tissue spaces of the body, and depends on the colloid osmotic pressure exerted by the plasma proteins. This varies directly

TABLE 5—SHOWING PLASMA VOLUME IN ML. PER KG. BODY WEIGHT IN RELATION TO ŒDEMA

Plasma volume ml./Kg.	*Normal Pregnancy*	*Œdema—Pre-eclampsia*			
	26 cases	*±* *6 cases*	*1+* *11 cases*	*2+* *12 cases*	*3+* *16 cases*
Maximum	52·8	51·4	51·2	48·0	44·5
Minimum	44·7	42·6	42·0	39·8	35·0
Average	49·4	47·1	45·7	43·3	38·8
Standard Deviation	3·22	3·73	3·08	2·80	2·35
Statistical Significance ..		+	+	+	+

with the concentration of the serum albumin, which has an osmotic tension of 6 mm. Hg. in a concentration of 1 gm. per 100 ml. The very large molecules of globulin and fibrinogen possess little osmotic property. The concentration of the total proteins in the plasma is of little value in the study of the relationship between the plasma protein level and œdema. That this is so is shown by the data provided in Table 6. The figures

TABLE 6—SHOWING PLASMA PROTEIN CONCENTRATION AND PLASMA ONCOTIC PRESSURE IN RELATION TO ŒDEMA

	Plasma Prot. Gm. %	Oncotic Pressure, cm. H_2O.
Normal Pregnancy	5·86±0·84	28·4±0·4 (28·9—27·8)
Hypertensive Toxæmia Oedema : 0	5·71±0·22 (6·20—5·41)	24·2±1·43 (27·5—21·7)
±	6·68±0·24 (6·16—5·20)	22·36±1·21 (24·5—21·8)
1+	5·87±0·27 (6·0—5·1)	27·4±1·92 (24·6—19·4)
2+	5·51±0·83 (6·14—4·95)	21·4±2·45 (24·7—18·1)
3+	5·17±0·80 (5·76—4·42)	20·2±1·96 (24·8—17·2)

The figures represent average±probable error, maximum and minimum values.

from which these data have been obtained were derived from 304 estimations on 100 cases of pre-eclampsia. Meticulous care was taken to eliminate cases of nephritis from this series, and all estimations were done in duplicate by the micro-Kjeldahl method. The figures marked under 'O' indicate values obtained from a series of 60 non-œdematous cases suffering from only hypertensive toxæmia with trace or no albuminuria.

The figures show that the total protein concentration of the plasma is slightly lower in the œdematous than in the non-œdematous hypertensive toxæmias. The difference in the values in the individual groups of œdema, however, is neither consistent nor statistically significant.

The importance of plasma proteins in œdema lies not in its total concentration, but in the colloid osmotic pressure they exert. This was estimated according to the formula provided by Wells and co-workers (1933) in all the cases mentioned above. These results are also presented in Table 6. It will be seen that the plasma oncotic pressure in the œdematous patients is slightly less than the value obtained in both normal and non-œdematous toxæmic pregnancy. This is true not only in the average value, but also for the range of values obtained in each series. In the œdematous group itself the differences in the colloid osmotic pressure are neither consistent nor statistically significant. The maximum values in each group show hardly any change while the œdema increases in severity.

Considerable literature has accumulated in recent years on the relation between hypoproteinæmia and the œdema of pregnancy toxæmia. The observations, however have not been unanimous. Thus, some observers (Zangemeister, 1916; Strauss, 1935, 1938) claim that the fall in the protein concentration of the plasma is responsible for œdema. Others (Stander, 1932; Tillman, 1939; Dieckmann, 1941) are of the opinion that the plasma protein level has no significant effect on the œdema. Dieckmann (1941) also calculated the oncotic pressure of the plasma in a small series of cases. He observed that the difference in the colloid osmotic pressure between the œdematous and non-œdematous toxæmic patients was only slight, and the magnitude of this was insufficient to explain the degree of œdema present. The data provided by Strauss (1938) are inconclusive, for some of his patients with hypoproteinæmia and œdema developed similar hypoproteinæmia but no œdema in the subsequent pregnancy.

Thus, the available evidences suggest that the alteration of the colloid osmotic pressure of the plasma is not the principal factor which decides the degree or the severity of œdema. However, it is probable that, hypoproteinæmia provides an additional factor in this condition, and acts by interfering with the reabsorption of fluids into the blood stream.

6. Capillary Permeability and Oedema

In 1916 Zangemeister suggested that an abnormal capillary permeability exists in toxæmia of pregnancy. Since then most writers appear to have resigned to this belief, for it provides an easy and satisfactory explanation for the œdema present in most cases of pre-eclampsia. Evidences in favour of such an hypothesis are, however scanty. The argument which is most often put forward in support of this theory is that the hæmatocrit reading and the serum protein concentration of the venous blood increase when the limb of a pre-eclamptic patient is subjected to gravity for some time. But, Youmans, Akeroyd and Frank (1935) have demonstrated that, even in normal individuals the serum protein concentration in the veins of the feet increase by 18 to 40 per cent under similar conditions. Dieckmann (1941) observed a 21 per cent increase of proteins in pre-eclamptic patients when subjected to these experiments. This hardly proves that the permeability of the capillaries in the inferior extremities of the body in pre-eclampsia is different from that of normal individuals. In view of the confusing nature of the evidences, it was considered that a study of the capillary permeability in pre-eclampsia may be interesting.

Capillary permeability was tested by the Gothlin petechial index in 86 cases of pre-eclampsia with moderate or marked œdema. The control study consisted of 44 cases of normal pregnancy, and 30 cases of hypertensive toxæmia of pregnancy without any œdema. In normal pregnancy the number of petechiæ varied from 0 to 13 with an average of $6{\cdot}2 \pm 1{\cdot}7$. In the hypertensive non-œdematous group the range of petechial index was between 2 and 18. Just over 60 per cent of cases had values of 8, or less than 8, the latter being the limit of normal capillary permeability. The average value in this group was $8{\cdot}1 \pm 2{\cdot}2$. In the œdematous patients suffering from hypertensive toxæmia of pregnancy the petechial index varied from 0 to 21, with an average of $8{\cdot}3 \pm 3{\cdot}8$. In 56·9 per cent of cases the value did not exceed the normal limit. In 39·1 per cent, the petechial index was between 9 and 12, which is regarded as the borderline zone. In only 4 per cent the value could be considered abnormal.

Griffith and Lindauer (1947) offered a new conception on capillary permeability, and described this condition as the rate at which the capillary endothelium permits fluid to pass through. Shanno, Griffith and LaMotte (1947) described a method using Patent blue by intradermal injection and measuring the spread of the dye streamers for a period of 15 minutes. The method was employed on those patients on whom the petechial index was studied. According to the accepted standards of normality (spread of less than ¾ inch from the site of the injection), normal pregnancy was attended with a slight increase of cutaneous lymph formation, and lymph flow. The spread in normal pregnancy was from $^{2}/_{3}$ to $1^{5}/_{12}$ inch, the average being 0·94 inch. In the non-œdematous toxæmic group the values obtained showed no deviation from this range. In only 6 out of 86 patients belonging to the œdematous group did the spread reach or exceed 1½ inches, and it should be mentioned that in none of these patients the œdema was marked. The calculated average value (0·95 inch) showed no noticeable difference from normality. The effect of gravity on the test, however, was interesting. When the limb was kept hanging, in normal pregnant women the streamers measured 1 to 1¼ inch. In non-œdematous toxæmic patients the values were almost identical, but in œdematous patients the spread exceeded the limit observed in normal pregnancy. The range of values was from 1½ to $2^{1}/_{12}$ inch.

Capillary permeability was tested by another method also. This consisted of a comparative study of the blood obtained from an arm vein before, and 30 minutes after the limb was allowed to hang alongside the edge of the bed. Before subjecting the limb to the effects of gravity it was maintained at rest at the heart-level for a period of half an hour. Throughout the experiment all forms of compression on the veins were avoided. Both samples of blood were examined for the packed-cell volume and plasma protein concentration. Tissue tension was measured both before and after hanging the limb. In addition, in the œdematous toxæmic patients tissue fluid was collected from the dorsum of the hand and examined quantitatively for total protein, chloride and sodium under basal conditions and after the arm had been subjected to gravity. The test was performed on 15 cases of pre-eclampsia and an equal number of normal pregnant women. The values obtained are shown in Table 7.

The figures demonstrate that the tissue tension, hæmatocrit and plasma proteins increased as a result of the experiment in both normal and toxæmic pregnancy. In normal pregnancy the increase in tissue tension was just over 5 per cent of the basic value, while in toxæmia this rise amounted to a little over 105 per cent over the initial level. The increase in hæmatocrit was equivalent to 9·1 per cent in normal pregnancy, and 17·4 per cent in toxæmia. The gain in the plasma protein concentration in normal pregnancy was by 11 per cent, and in toxæmia by 16·5 per cent. The increase in the hæmatocrit readings and the plasma protein concentration, allowing for experimental errors, are comparable in each group. These findings, together with an increase in tissue tension caused by gravity point to an increased diffusion of fluid (or, a retarded absorption) into the tissue spaces, which is present in normal pregnancy, but considerably augmented in toxæmia. The concentration of proteins in the tissue fluid throughout the whole experiment show hardly any alteration in spite of the changes in the tissue pressure, and the composition of the blood. Had the capillaries been abnormally permeable in toxæmia of pregnancy it would have been

TABLE 7—SHOWING THE EFFECT OF GRAVITY ON THE PERMEABILITY OF THE CAPILLARY ENDOTHELIUM

		Hæmatocrit		*Pl. Prot.*		*T. Tensión*		*T.F. Prot.*		*T.F. Chlor.*		*T.F. Na.*	
		1	*2*	*1*	*2*	*1*	*2*	*1*	*2*	*1*	*2*	*1*	*2*
Normal pregnancy	Average ..	31·2	34·3	6·36	7·06	17·7	27·0	—	—	—	—	—	—
	S.D. ..	0·81	1·34	0·35	0·48	2·3	1·7	—	—	—	—	—	—
	Minimum ..	29·9	32·4	5·82	6·24	14·0	23·5	—	—	—	—	—	—
	Maximum ..	33·0	37·1	6·90	7·67	21·6	30·0	—	—	—	—	—	—
Pre-eclampsia (œdema)	Average ..	33·9	39·8	5·39	6·33	27·4	56·5	0·16	0·17	598	599	318	330
	S.D. ..	1·2	0·77	0·34	0·37	3·3	5·2	0·04	0·03	8·91	8·62	13·4	17·5
	Minimum .	31·9	38·5	4·90	5·77	23·8	49·8	0·10	0·10	585	585	308	315
	Maximum ..	35·6	40·9	5·80	6·85	34·2	64·0	0·21	0·20	609	609	346	360

1—Basic Values. 2—Values at the end of the experiment

natural to expect a rise in the protein content of the tissue fluid under the conditions of the experiment. The protein content of the œdema fluid is always low, and in this respect these findings are in close agreement with those of other investigators (Dexter and Weiss, 1941; Albano, 1927; Hellmuth, 1922). These facts suggest that the function of the capillary endothelium as a semipermeable membrane is not in any way affected in toxæmia of pregnancy. The permeability of the capillaries is not increased, although the diffusion of fluid across the capillary membrane increases considerably. This is governed not by any alteration of the physical character of the cells, and their cement substance separating the intra-vascular from the extra-vascular spaces of the body, but by a physico-chemical process, the manifestation of which is the increase in the concentration of sodium in the tissue fluid caused by a retarded venous return induced by gravity. The significance of the retention of sodium in the tissue fluid in the alteration of the electrolytic equilibrium and the production of œdema has been demonstrated already in a previous communication (Mukherjee and Govan, 1950).

SUMMARY AND CONCLUSION

The results of an investigation into the causes of œdema in pre-eclampsia are presented. The relation between œdema and the following factors was studied: hypertension, albuminuria, oliguria, plasma volume, plasma proteins and capillary permeability. It is concluded that none of these physical factors can by themselves give rise to œdema in pregnancy toxæmia. In presence of the electrolytic inequilibrium (retention of sodium, organic acids and phosphates) which has previously been demonstrated to be the cause of œdema, these factors may play an auxiliary rôle. Hypertension is of little importance, albuminuria acts by depleting the plasma of its protein content, and this causes a reduction of the oncotic pressure. The oliguria may be partly an effect of the pre-renal deviation of the water into the tissue spaces. This is borne out by the studies on the plasma volume and hæmatocrit. There is no evidence of damage to the capillary endothelium caused by the widely believed toxin of toxæmia of pregnancy.

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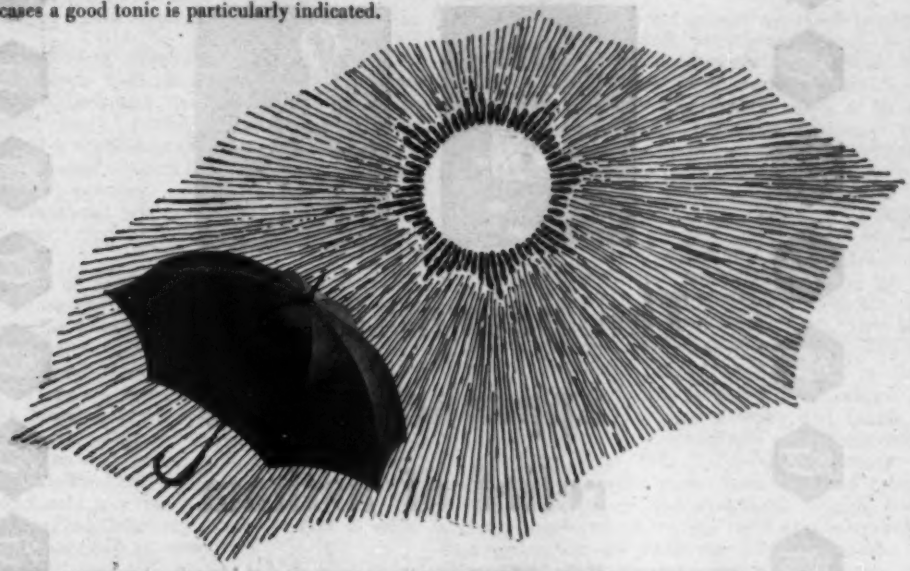

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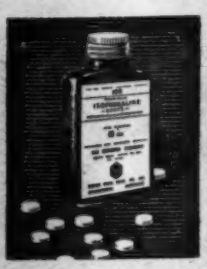

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ACUTE ASCENDING MYELITIS FOLLOWING THE ADMINISTRATION OF NEOARSPHENAMINE

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Acute ascending myelitis, producing an acute ascending paralysis associated with sensory changes and sphincter disturbances, has been described in association with many pathological conditions. The causative agent is not always the same. Wortis (1947) described this disease to occur in the course of influenza, poliomyelitis, pregnancy, gonorrhœa, chicken-pox, measles, streptococcal infections, typhoid fever, rabies, undulant fever, pyelonephritis, tooth abscess and infected tonsils.

The rarity of acute ascending myelitis following arsenicals is brought out by the fact that most text books do not list it as one of the toxic manifestations of neoarsphenamine therapy. Scott and Rheinhart (1930) and Moseley and Callaway (1940) recorded acute diffuse myelitis, after intravenous neoarsphenamine. Two cases of acute myelitis following injections of acetylarsan were reported by Gadrat (1946). Arsenical myelitis in syphilitics has been recorded by Paraire and Hosotte (1947). Blankenhorn and Wolff (1948) found only nine cases in literature similar to the case of myelitis following administration of neoarsphenamine described by them.

The case reports are so few that no clear cut clinical picture could be described as typical of arsenical acute ascending myelitis. The histopathological changes in the cord similar to that in the brains of patients with encephalitis and in the brains and cords of encephalomyelitis have been described in the still fewer autopsies available.

The following case is recorded because of the rarity of the accident and the few points of interest in the onset, progression and recovery of the condition.

Case Report

S.S., a Hindu male, aged 30 years was admitted to the Sarojini Naidu Hospital, Agra on 20-7-1950. He complained of inability to use both lower limbs and left arm, and a sensation of numbness, duration 20 days.

He had developed a sore on penis overnight after a doubtful exposure to syphilis. He was given a course of ten intravenous injections of neoarsphenamine, by his doctor, during a period of three months before admission. On the day following the tenth injection, 20 days before admission, he felt numbness in the right leg and did not feel the sensation of cold in the right foot and leg below the knee when he washed his hands and feet with ice water after the day's work in a glass factory. Within a week of onset of these symptoms he felt weakness in both legs below the knees and inability to appreciate cold. This was followed by inability to walk. By the end of the second week, a sensation of numbness rose up the patient's leg to the trunk and arms. A feeling of heaviness and loss of sensations was felt in the left upper limb, followed two days later by inability to lift the arm. Heaviness in the right arm was experienced on the day previous to admission.

On examination the patient appeared moderately ill. His memory and intelligence were normal. Cranial nerves showed no abnormality. Both lower limbs and left upper limb showed complete loss of motor power, whereas the muscular power of the right arm was diminished. All the four limbs were hypotonic but there was no wasting and no fibrillary twitchings. There was complete sensory loss over both lower limbs, left upper limb, abdomen and chest. The sensations of touch, pain and temperature were impaired on the right arm and normal over the head, face, neck and upper part of the chest. The deep reflexes were diminished in the right arm and lost in the left arm and both lower limbs. Abdominal reflexes were absent and plantar reflexes indefinite. The heart, lungs and abdomen showed no abnormality. The temperature was normal.

On 22-7-1950, two days after admission, the patient developed retention of urine and fæces. He had to be catheterised 8-hourly and enema was ordered on alternate days. On 25-7-1950 the patient was unable to lift his right upper limb. He had complete loss of motor power and sensations. Biceps, triceps and supinator reflexes of the right arm were absent. The total white cell count was 6800 per c.mm. The differential count showed neutrophils 58 per cent, lymphocytes 26 per cent, eosinophils 15 per cent and monocytes 1 per cent. Cerebrospinal fluid was under normal pressure, cells 36 per c.mm., mostly lymphocytes, sugar 59 mgm. per cent, chlorides 750 mgm. per cent and proteins in fair amount. Wassermann reactions of both blood and cerebrospinal fluid were negative. The urine showed no abnormality.

On 26-7-1950, at 4 p.m. he suddenly became markedly dyspnoeic and looked actutely ill. The abdomen did not move with respiration and paralysis of the diaphragm was suspected. Intercostal muscles were working feebly. The anaesthesia had extended upto the neck. All the jerks were lost and plantars indefinite. He was put on oxygen inhalations and stimulant line of treatment. Iron lung could not be used as it was not in working order. On 27-7-1950, he was less dyspnoeic and diaphragmatic movements could be seen. By 29-7-1950, the dyspnœa had disappeared and the diaphragm and intercostal muscles were working normally.

On 7-8-1950, the first evidence of regression of the level of anæsthesia appeared. Touch, pain and temperature sensations were now absent below the manubrium sterni. On 22-8-1950, the level of complete anæsthesia receded to the level of the sixth rib on the right side of the chest and fourth rib on the left side. Diminished sensations were recorded in both upper limbs, and feeble biceps, triceps and supinator

reflexes could be elicited. Cerebrospinal fluid was re-examined. It was under normal pressure, cells mostly lymphocytes, sugar 45·4 mgm. per cent, chlorides 740 mgm. per cent, proteins in fair amount. Wassermann reactions in blood and cerebrospinal fluid were completely negative.

On 30-8-1950, the sensations returned to normal above the level of ninth rib on the right side and sixth rib on the left side. On 10-9-1950, the level of anæsthesia was at the lower costal margin. Biceps, triceps and supinator jerks were exaggerated on both sides. On 20-9-1950, the level of sensory loss was at the umbilicus. Plantar reflexes on both sides were extensor. On 15-10-1950, all sensations returned to normal. There was no patch of anaesthesia. Ankle jerks were exaggerated. On 20-10-1950, knee jerks were exaggerated. Patient had full control over urine and faeces, catheterisation was no more necessary. The patient was able to sit up in bed. On 30-10-1950, the patient was able to walk with support. He developed bronchopneumonia the same evening. Total white cell count rose to 19,500 per c.mm. Differential cell count showed neutrophils 95 per cent and lymphocytes 5 per cent. He responded to pencillin therapy.

On 26-12-1950 he was examined for a complete check up before discharge. Intelligence and memory normal. Cranial nerves showed no abnormality. All sensations were normal. Motor power normal in arms and hands, but slightly diminished in both lower limbs with a little increase in tone. Biceps, triceps, supinator, knee and ankle jerks were exaggerated on both sides but equal. Babinski reflex positive on both sides. Abdominal reflexes absent. Gait slightly spastic. Total white cell count 8500 per c.mm.; neutrophils 76 per cent, lymphocytes 23 per cent, eosinophils 1 per cent. Total red cell count 4,450,000 per c.mm.; hæmoglobin 12 gm.; sedimentation rate 11 mm. in 1 hour. Cerebrospinal fluid under normal tension, cells 27 per c.mm. mostly lymphocytes, sugar 50 mgm. per cent, chlorides 760 mgm. per cent, proteins in traces. Wassermann reaction negative in both blood and cerebrospinal fluid.

Discussion

The abrupt onset of the disease; the appearance of sensory loss in the legs and its upward spread to the trunk, arms and neck in that order; complete motor paralysis proceeding to involve the muscles in the order of their innervation from the spinal cord; followed later with increased reflexes and extensor plantar reflexes; and the loss of sphincter control left us in no doubt about the diagnosis of acute ascending myelitis. Landry's paralysis and acute ascending poliomyelitis were considered at the time of admission but were ruled out on account of sensory disturbances and involvement of the sphincters. Absence of complete recovery or residual persistent atrophic paralysis typical of surviving patient only confirmed our earlier impressions. The sensory loss on the trunk, the absence of abdominal reflexes, the involvement of sphincters, and the appearance of spastic paraplegia with increased knee and ankle jerks and extensor plantar were against the diagnosis of the acute ascending polyneuritis. Thecal hæmorrhage is accompanied by ascending paralysis, severe sensory loss and sphincter disturbances, but in the absence of blood in the cerebrospinal fluid and the comparatively slow progression of the disease, this diagnosis could not stand.

That syphilis was not the cause of myelitis is evident from the repeatedly negative serological tests of the blood and cerebrospinal fluid and uncertain history of exposure to the infection. The chance occurrence of myelitis of unknown etiology in a patient receiving arsenicals cannot be altogether disregarded. That the therapeutic use of arsenic may produce several central nervous system reactions—encephalitis, encephalomyelitis and myelitis is well recognised. Scott and Rheinhart (1930) and Blankenhorn and Wolff (1948) described identical cases, in which acute myelitis followed the second injection of neoarsphenamine. The symptoms appeared after the tenth intravenous injection of neoarsphenamine in the case reported above.

The disease is usually rapidly fatal, many cases succumb in the acute stage because of respiratory involvement. In view of successful use of BAL by Eagle and Magnuson (1946) in 55 cases of arsenical encephalitis and by Blankenhorn and Wolff (1948) in acute arsenical myelitis, the therapeutic trial of this drug was considered but it was not available. Apart from the symptomatic treatment and Vitamin B_1 100 mgm. daily intramuscularly for the first four weeks since admission to the hospital, he had no other treatment. The recovery was, therefore, considered spontaneous. This again opens the question whether myelitis, or for that matter every other central nervous system reaction, following the injection of neoarsphenamine is due to toxic effects of this injection or an allergic manifestation to the drug.

Summary

Arsenical therapy as the cause of acute ascending myelitis has been discussed, and supported by a case record and review of the literature.

Acknowledgment

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PULMONARY AMŒBIASIS

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Burdwan

Introduction

Amœbiasis is prevalent very widely in our country, being one of the common causes of morbidity. This is specially so in the District of Burdwan in the province of West Bengal. In amœbiasis, manifestations are mainly intestinal; dysenteric or irregular diarrhoeic motions, alternation of constipation with diarrhoea, or in late stages, constipation with dyspepsia and in some cases vague abdominal discomfort with lassitude and hypochondriac features are the commonest complaints. Hepatic complications with or without abscess formation come next in frequency. Till recently pulmonary complications were considered rare and more of academic interest than of any practical significance. Within the last five years, thoracic complications of amœbiasis have received more attention than previously. Thus Chaudhuri (1946) reported two cases of pulmonary amœbiasis. Chatterji (1949) reported a case of multiple lung abscess. A case of putrid lung abscess was reported by Mukherji (1949). Two cases of amœbic lung abscess, diagnosed post mortem, were placed on record by Balbir Singh (1949). Ten cases were reported by Koszalka in 1949. Bookless (1950) referred to ten cases of atypical chest syndromes in patients suffering from amœbic hepatitis. Mention has been made by Manson-Bahr in his book on tropical medicine (1931) and Dormer and Friedlander (1941). Rogers (1935) found secondary lung abscess in 26 patients out of 81 liver abscess cases in autopsy.

Pathogenesis

The exact mode of invasion of lung tissue by *Ent. histolytica* is not clear and is at best speculative. In the presence of hepatic amœbiasis, with or without abscess formation, it has been suggested that the lung may be involved:—(*a*) by a process of contiguous spread through diaphragm, (*b*) by transdiaphragmatic lymphatic spread and (*c*) by venoarterial circulatory carriage. *Ent. histolytica* has been thought to enter the tributaries of hepatic veins, to be carried to the right side of the heart, thence through pulmonary artery and its branches to the lung field. The mode of pulmonary invasions by *Ent. histolytica* in the absence of obvious hepatic amœbiasis (so-called primary pulmonary amœbiasis) is still more obscure and embolism has been thought to be the cause.

Clinical Varieties

Clinically, pulmonary amœbiasis may be of the following types:—

(a) *Symptomatic.* In this group there is no involvement of lung tissue radiologically. Main features are complaints of pain in the shoulder region or lower part of chest, mild-grade pyrexia with irritable cough. Pleural rub may be present. X-ray reveals a raised dome of diaphragm with or without a small effusion and a restricted mobility of the diaphragm may be seen on fluoroscopy. Clinical features appear to be due to involvement of the diaphragmatic pleura, which is supplied by phrenic nerve (C 3-5) and lower six intercostal nerves. According to some workers, 15-25 per cent of patients suffering from amœbic hepatitis, have been found to present chest complaints, without obvious pulmonary involvement. In such cases, with emetine therapy, there is complete amelioration of thoracic complaints, as also disappearance of pleural rub, when preesnt, and rearogression of the liver.

(b) *Secondary Pulmonary Amœbiasis.* In this group of patients there is actual invasion of lung tissue from infected liver by a process stated earlier. *Ent. histolytica* on entry, causes cytolytic necrosis of lung tissue which may be infected by secondary organisms later on, giving rise to signs resembling basal pneumonitis situated close to the diaphragm whose dome may be raised and merging with the opacity of the lung. The opacity usually tails off towards the hilum. With the establishment of communication through bronchus or in the event of partial absorption of necrotic tissue, the feature of an abscess cavity is established clinically and also radiologically. A fresh specimen of sputum in such cases may show motile form of *Ent. histolytica.* Pus cells may be present indicating secondary infection: pleural effusion may occur, a clear fluid indicating sympathetic effusion and purulent fluid indicating actual invasion by *Ent. histolytica.* Unless pus accumulates in pleural cavity in some quantity, response to emetine therapy with penicillin administration parenterally for secondary infection is quite satisfactory.

(c) *Primary Pulmonary Amœbiasis.* It means amœbic pulmonary involvement, without obvious amœbic hepatitis. This is definitely rare. Radiologically, like secondary pulmonary amœbiasis, it may present the picture of (*a*) a patch of pneumonitis (clinically, closely resembling pulmonary tuberculosis) and (*b*) a necrotic abscess cavity, to be differentiated from a putrid lung abscess.

The author came across six cases of pulmonary amœbiasis recently. Out of these six, three were admitted during his stay as teacher of medicine and physician-in-charge of Fraser Hospital, Burdwan. The other three cases were treated outside the hospital. The case reports are given below.

Case Reports

Case 1—S. P. Nayek H. M., aged 20 years, reported for treatment during early part of March 1949, for pain in the right side of his chest and back, cough with thick expectoration, a feeling of malaise and tiredness towards afternoon and apparent loss of weight. History revealed that he was a chronic sufferer of dysentery and had mild-grade hepatitis for a long time. He had chronic malaria and rheumatic mitral stenosis without objective cardiac failure. Recently, however, he was having subjective cardiac symptoms on exertion and had to be kept on a maintenance dose of digitalis after full digitalisation.

On examination, he was found to be slightly anæmic. Spleen—palpable 3 fingers below the costal margin and firm; Liver—palpable 2 fingers below the costal arch and tender; cæcal thickening, without tenderness—present; lungs—few scattered rhonchi, expiration slightly prolonged in general and air entry deficient at lower zone posteriorly, without any adventitious sound. Heart revealed evidence of old-standing mitral stenosis (pulmonary systolic murmur in addition to presystolic in the mitral area). No enlargement of the heart clinically discernible. *Laboratory findings*—Blood count: W.B.C.—8,600/c. mm.; poly—50 per cent, lympho—41 per cent, mono—2 per cent, eosino—7 per cent. Skiagram of the chest—(10-3-49) revealed a fairly big homogeneous opacity, contiguous to the right dome of the diaphragm, which showed central tenting; the appearance was highly suggestive of a pneumonic process having a transdiaphragmatic connection with the liver. Sputum was sent for examination which revealed motile *Ent. histolytica.* This clinched the diagnosis. Besides symptomatic treatment and digitalization, he was put on a course of emetine therapy, under a condition of perfect bed rest. He had a total of 6 grains of emetine. Skiagram of chest taken after the completion of 6 grains of emetine showed practical disappearance of basal pneumonitic patch, only the tenting of diaphragm remaining. Clinically also he became free from chest symptoms (Figs. 1A and B, *vide* Plate).

Case 2—A. R. was admitted in the Fraser Hospital on 3-11-48 as an observation case for hæmoptysis. He, a chronic sufferer of dysentery and mild-grade hepatitis was having cough with thick slightly yellowish expectoration, towards early morning and malaise towards evening for about a month. On the day before admission he had streaks of blood in his sputum, repeated several times.

Examination—anæmic, asthenic, liver one finger enlarged and tender; caecal thickening present. Diminished air entry on the right side of chest, lower zone posteriorly, no adventitious sound. Blood: slight leucocytosis; sputum—no a.f.b., pus cells present, r.b.c. present; X-Ray of the chest showed a basal opacity just adjoining central portion of the diaphragm with bronchovascular shadow prominent on right side (Fig. 2A *vide* Plate). After the hæmoptysis was controlled, sputum was sent for examination again. Report read "No acid-fast bacillus, few motile *Ent. histolytica* present". This led us to try emetine grain 1, I.M. daily for 6 days. This controlled his fever and cough: expectoration ceased. After a rest period of 3 days, he was given 3 grains more of emetine. Skiagram taken after this (18-11-48) showed disappearance of the homogeneous opacity (Fig. 2B, *vide* Plate).

Case 3—Gati, H. M., aged 48 years, was admitted in the Fraser Hospital in the first week of November 1948, with a history of illness of two week's duration. He complained of vague bodily discomfort including chest aches, cough with scanty expectoration and slight breathlessness. Spleen—palpable 4 fingers below the costal arch; Aldehyde test—negative; Widal—negative. Urine and stool showed no abnormality. Liver—palpable 2 fingers below the costal margin and tender. Blood: moderate leucocytosis (W.B.C. 10,600/c.mm.) with polycytosis (poly 86 per cent). Sputum—pus cells in fair number. Expiration—in general slightly prolonged; rhonchi—scattered, rales—few, right midzone, posteriorly; Breath sound—deficient with impaired resonance at right base. Clinical picture together with blood count suggested a patchy pneumonitis at right base or a small effusion. He was advised penicillin 1 lac 3 hourly. Even after 24 lacs, however, clinical condition remained uncontrolled. Skiagram of chest taken on 20-11-48 (Fig. 3 *vide* Plate) showed pleural effusion on the right side and fibrotic scar marks in the right upper zone. The fluid was tapped for diagnosis. It was clear; microscopically there was no R.B.C. or pus cell. Though no active tubercular lesion was radiologically detectable, streptomycin was advised because of pleural effusion, chest complaints and fever. Even after 8 gm. of streptomycin, however, there was no abatement of the condition in any way. His breathlessness increased, fever persisted and cough became more and more productive and was of slightly foul odour.

The persistence of the clinical condition of the chest, resisting known chemotherapeutics and antibiotics (he had irregular administration of sulphadiazine also before admission), the basal nature of clinical manifestation on the right side of the chest with association of tender liver and the demonstration of *Ent. histolytica* in the sputum of two other cases in the same hospital, led us to search for *Ent. histolytica* several times a day from fresh specimens of sputum. After repeated search, the pathologist was able to demonstrate a few *Ent. histolytica* in the sputum. This clinched the diagnosis. Therapeutic response was dramatic with three emetine injections. He was given 9 grains of emetine before he was discharged fully cured from the hospital.

Case 4—K. A. S., male adult, reported for treatment on 29-1-1951 for pain on the right side of the chest with cough and scanty expectoration lasting 3 weeks. No history of fever or hæmoptysis. Past history of repeated attacks of dysentery with pain in the right hypochondrium off and on. Examination showed the patient to be an old man, apparently healthy. The liver was enlarged three fingers and slightly tender; caecal thickening or tenderness absent; lungs—the right lower zone showed impairment of resonance with deficient air entry, fine crepitations on deep inspiration over the right base posteriorly; sputum showed no acid-fast bacillus, but a fair number of pus cells were present. Blood count: total W.B.C.—6,800/c. mm.; poly—67 per cent, lympho—26 per cent, mono—2 per cent, cosino—5 per cent. Skiagram of the chest on 29-1-1951 showed very soft homogeneous opacity with central small cavitation on the right base with patchy small opacity in the lower part of the right midzone, with exaggerated bronchovascular marking on the right side. The dome of the diaphragm

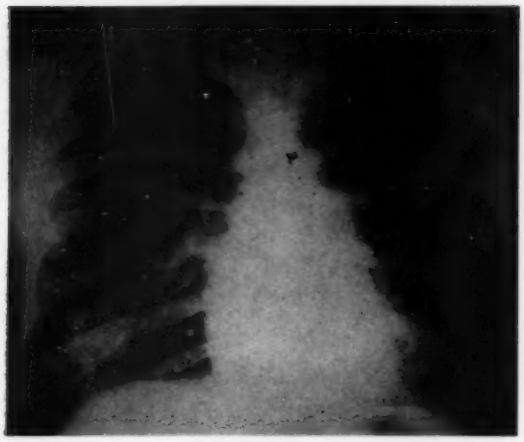

FIG. 1A (Case 1)

FIG. 1B (Case 1)

FIG. 2A (Case 2)

FIG. 2B (Case 2)

FIG. 3 (Case 3)

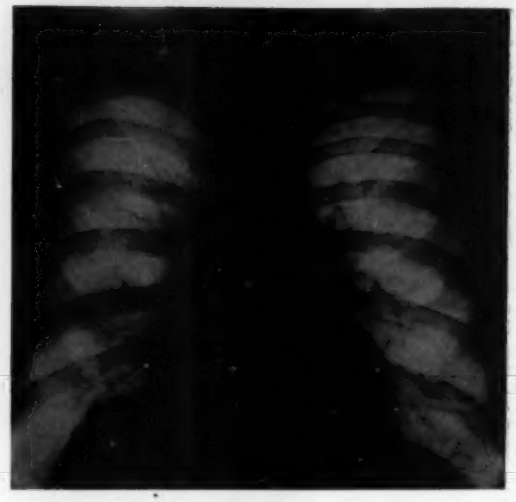

FIG. 4A (Case 4)

FIG. 4B (Case 4)

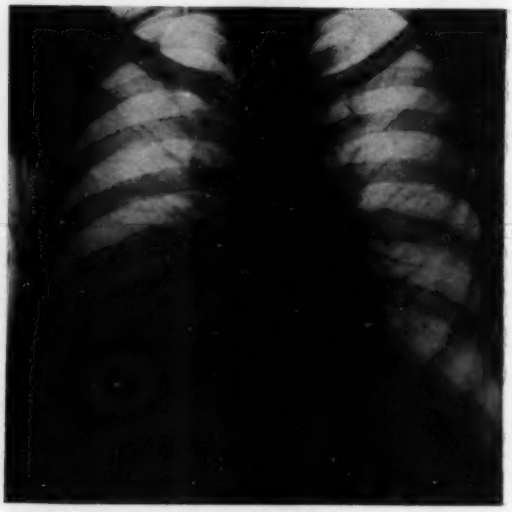

FIG. 5A (Case 5)

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on the right side was elevated. The short history of chest pain on the right side with tender liver, raised dome of the diaphragm and basal opacity led us to try emetine grain 1 I.M. daily for 6 days, then grain 1 daily for 3 days more after 3 days' rest. Except mild chest pain and dry cough, there was no complaint. Expectoration ceased, the liver became 1 finger palpable and was still slightly tender. Skiagram on 22-2-1951 showed disappearance of almost the whole of the homogeneous opacity except exaggerated bronchovascular marking and slight peribronchial opacity. The patient was advised to take 3 grains more of emetine (Figs. 4A and B, *vide* Plate).

Case 5—S. G., Hindu male, aged 42 years. History of passing dysenteric stools, lasting 5-6 months, irregularly treated, reported for treatment on 15-2-51 for irregular low grade fever with bouts of cough with highly offensive sputum of a fishy odour; quantity of the sputum expectorated was roughly ½ to 1 seer a day. Lately, he was having profuse expectoration, which looked like a mixture of blood and pus. He also had pain on the right side of the chest and the abdomen. On examination he was found to be pale, anæmic and exhausted. The liver was palpable 3 fingers below the costal arch and was tender. Intercostal tenderness was present but there was no surface œdema. Chest examination revealed on the right side dullness in the midclavicular line fourth space downwards and midaxillary region fifth space downwards; breath sound very much diminished in intensity in the right lower zone. Blood count: W.B.C.—7,200/c.mm.; poly—72 per cent, lympho—18 per cent, mono—3 per cent, eosino—7 per cent. Skiagram of chest on 15-2-51 showed a fairly big cavity with a fluid level and with a broad wall in the right base. The sputum on examination showed no *Ent. histolytica*, but plenty of pus cells. Absence of high leucocytosis as is to be expected in a case of pyogenic lung abscess, presence of a highly tender liver in proximity and the nature of the sputum and the fact that in the same month we had treated another case of pulmonary amœbiasis successfully, led us to try emetine in this case also. He was given procaine penicillin sodium, 4 lac units on the first day and from next day onwards he was ordered only emetine—grain 1 I.M. daily for 6 days and then grains 6 more after a rest period of 3 days. Expectoration completely ceased after 4 grains of emetine and except for persistence of mild pain on the chest on deep inspiration, he had no complaint after 12 grains of emetine. The liver became reduced in size to 1 finger below costal magin and was not tender. Another skiagram of the chest on 5-3-51 showed disappearance of almost the whole of the radio-opacity, excepting a thinned out narrowed area indicating the upper zone of the former abscess cavity (Figs. 5A and B, *vide* Plate).

Case 6—S. C. adult female came on 26-11-50 complaining of mild-grade pyrexia towards evening daily, associated with irritable cough and scanty expectoration for about a month and a half. For a long time she was keeping good health except as she said for a bout of indigestion and loose motion for a temporary phase of about a week, six months ago, which was controlled by her attending doctor with 6 grains of emetine. Lately, however, she was having an indifferent health with progressive lassitude, weakness and loss of appetite. The feverish feeling towards evening daily with cough unnerved her and that brought her to seek medical advice. On examination she was found to have a good general health, no obvious septic focus was detected. The spleen was not palpable. The lungs clinically appeared to be free from abnormality. Heart—N.A.D. The liver was just palpable and not tender. As no obvious cause was discernible, a skiagram of the chest was taken as a first step on 8-12-50 (Fig. 6, *vide* Plate). It showed a small rounded opacity in infraclavicular zone peripherally and a mottled shadow on left midzone. There was no eosinophilia on blood count. A provisional diagnosis of an early pulmonary tuberculosis was made and the patient was advised to keep a weight chart and temperature chart and to report again after six weeks for a fresh skiagram of the chest. Three days later, however, the patients' husband came complaining that the patient was having distressing cough and each time she was expectorating a highly offensive brownish liquid sputum, though small in quantity, the odour was particularly annoying to her. She was advised to bring the sputum. On examination it was found to be resembling exactly a dysenteric stool. It was unbelievable that it could be sputum. So he was avised to bring the patient. On coughing she expectorated sputum of the very same character. Microscopically, it showed plenty of *Ent. histolytica* with ingested R.B.C. She was advised emetine grains 9. After completion of the injections, the character of her sputum changed, the expectoration practically ceased, but her original complaint of mild-grade pyrexia and loss of weight is still persisting. She is being investigated.

Summary

Report of six cases of pulmonary amœbiasis has been submitted. These six cases have been collected during the last two years.

In the first two cases, diagnosis was confirmed by demonstration of *Ent. histolytica* in the sputum.

Case 3 presented difficulty in clinical diagnosis, credit goes to the pathologist for his persistent search and ultimate demonstration of motile form of amœba. The pleural effusion perhaps masked the underlying lung involvement in x-ray.

Diagnosis in Case 4 had therapeutic and radiological corroboration.

Similarly, in Case 5 a cavity resembling a putrid lung abscess was seen in the x-ray.

Case 6 is difficult of interpretation. The sputum definitely showed *Ent. histolytica* and change in its character occuring with emetine, but its source was not evident. The dome of the diaphragm throughout was clear and nowhere, could any pointing or

humping be seen. On the other hand, there was no indication of abscess cavity in the lung also radiologically. The infiltrational lesion in the subapical zone (right) and left midzone can hardly be thought of, to be of amœbic origin. These are probably of tubercular origin and are responsible for keeping up the fever and mild cough even now. A serial skiagram of chest from different angles which the party could not afford might have shown a small cavity in the lung field. In the absence of information and further investigation, the source of *Ent. histolytica* in sputum in this case remains obscure.

Conclusions

To conclude, in patients suffering from amœbic hepatitis presenting chest symptoms, *e.g.*, fever with pain chest, cough with expectoration of foul sputum or blood-tinged sputum, diagnosis of pulmonary amœbiasis should be thought of.

In the event of the picture not fitting in with the diagnosis of pulmonary tuberculosis or pyogenic or putrid lung abscess, the sputum should be searched for *Ent. histolytica* in fresh specimen, presence of which will at once clinch the diagnosis. Even in its absence, a basal homogeneous opacity or a basal cavity specially in the right side not responding to usual treatment, justifies the therapy with a course of emetine. Clinical amelioration with radiological disappearance of lesion, justifies the diagnosis of pulmonary amœbiasis indirectly.

In pulmonary amœbiasis diagnosis the examination of stool has a very limited place. It may be suggestive but not diagnostic. For in many cases stool examination may be negative for *Ent. histolytica*. On the other hand, their presence do not vouchsafe for the pulmonary lesion, to be amœbic in origin.

Acknowledgment

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PSEUDOHERMAPHRODITISM

With Reports of Two Cases

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Hermaphroditism is an old term that originated with the ancient Greeks. It means a combination of masculine and feminine characters in the same individual. Hermaphroditism may be of two types, true and false.

True hermaphroditism is extremely rare. In the strictest sense a true hermaphrodite should have functioning gonads of both sexes and should be able to function both as father and as mother. No such case has been reported. But using the term in a narrower sense, individuals possessing both testis and ovary, or ovotestis, are usually classed as true hermaphrodites. In 1915, Pick collected 4 cases of ovotestis. In 1937 Young collected 20 authentic cases. In 1944, Gudernatsch collected 37 such cases from the literature.

False or pseudohermaphroditism exists when the gonads are of one sex but the external genitals have some features of the opposite sex. This is also rare, though more common than true hermaphroditism. Neugebauer in 1908 collected 942 such cases but most of them lacked scientific details and only in 41, the sex was definitely established. Pseudohermaphrodites are named according to the sex of the gonads. Individuals possessing testes are male pseudohermaphrodites and those with ovaries are female pseudohermaphrodites. Male pseudohermaphroditism is more common.

Formerly hermaphrodites were looked upon as so many curiosities or as sexual monsters having a combination of male and female characters assembled without any rhyme or reason. But the development of modern science such as biology, embryology, endocrinology and genetics has led to a better understanding of this subject.

From a study of embryology we know that the early human embryo is potentially a hermaphrodite. Upto the 7th week of fœtal life, the gonads and external genitals have not differentiated and both the excretory ducts, the wolffian and the mullerjan, develop in every embryo. After the 7th week a normal fœtus develops its sex organs only in one direction. It is now generally accepted that the sex of the fœtus is determined by the chromosomes at the time of fertilisation. It has been shown (in lower animals) that this chromosomal determination of sex may be modified or reversed by other factors such as hormones, diet or temperature (Goldsmidt, Greene, Zuckerman, Shepperd and others). From such experiments on butterflies Goldsmidt first developed the modern theory of *intersexuality*. Moszkowicz of Vienna further developed this theory as applied to human pathology.

"By an intersex they refer to such individuals who started out either as a male or as a female from a

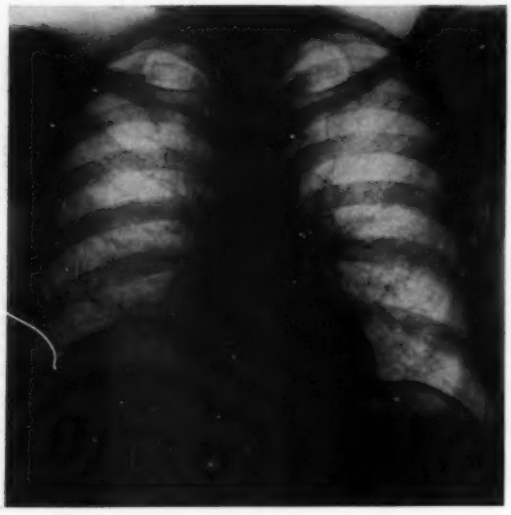

FIG. 5B (Case 5)

FIG. 6 (Case 6)

FIG. 1—Male pseudohermaphrodite. Note the development of breasts (Case 1).

FIG. 2—Male pseudohermaphrodite. External genitals before operation (Case 1).

FIG. 3—External genitals after operation (Case 1).

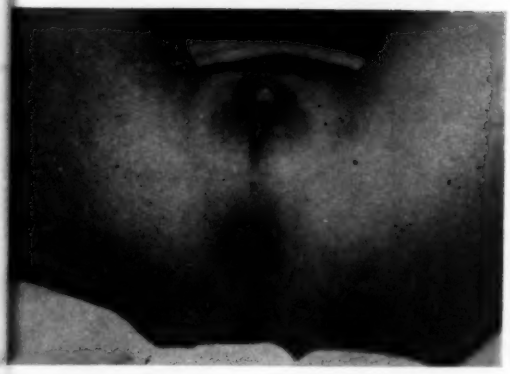

FIG. 4—Female pseudohermaphrodite. External genitals before operation (Case 2).

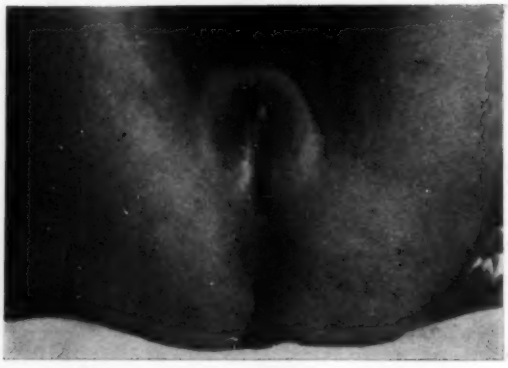

FIG. 5—External genitals after operation (Case 2).

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genetic point of view but after sometime completed their development in the opposite direction resulting in the mixture of opposite sexual characters in the same individual. The time at which this reversal takes place is called the *rotation point*. Each intersex is sufficiently characterised by its primary sex until the rotation takes place. Moszkowicz prepared detailed diagrams demonstrating the normal development for each month of fœtal life, so that by a careful study of the intersex the *rotation point* can be explained." (Schiller).

Goldsmidt insisted that the human intersexes were always primarily female and secondarily male. Majority of the intersexes are male pseudohermaphrodites and in these cases the primary sex is female and the gonads which were destined to be ovaries are completely replaced by testicular tissues. In order to apply Goldsmidt's theory to the female pseudohermaphrodites we have to imagine *two rotations* during the development of the fœtus. The individuals start as females, for some time develop as males, but finally complete their development as females.

Broster and Vines have demonstrated in the cortical tissue of adrenals (between the 9th and 17th weeks of fœtal life) the same fuchsinophil cells which they found in cortical hyperplasia of adolescent virilism. These cells are found both in the male and in the female embryos but in the male they are more abundant and last longer than in the females. They believe that the degree of masculinisation in hermaphrodites depend on the abundance and persistence of these cells.

Sex organs and sex characters in pseudohermaphrodites may show a wide range of variation. Theoretically it is possible to have innumerable varieties of combinations of male and female characters and in the reported cases we may find nearly as many varieties as there are cases. They may, however, be summarised in general terms as follows:—

(1) Gonads are testes in the male and ovaries in the female. They are not always in their normal place. The testes have been found in the abdomen, in the groin or in the pelvis. The ovaries may be found in the abdomen, in the inguinal canal or in the labia.

(2) The wolffian and the mullerian ducts—in the normal individuals only one of these develops and the other atrophies and remain as vestigial structure. In pseudohermaphrodites both may be atrophied; or partial development of one may be associated with non-development or deficient development of the other; or one or the other may be almost normally developed.

(3) External genitals in a male pseudohermaphrodite are generally feminine. But the clitoris is usually enlarged simulating hypospadiac penis and the vagina is usually rudimentary. In a female pseudohermaphrodite the external genitals are somewhat masculine. The enlarged clitoris looking like a hypospadiac penis and the labia minora showing varying degrees of union, sometimes even forming a clitoridian urethra.

Along with these there are atypical combinations of secondary sex characters, bodily configuration and of mental outlook.

In order to explain such atypical combinations, Wolff of the University of Bern suggested that the primary sex makes a permanent impress on the somatic and psychological cells prior to the development of the gonads.

The diagnosis of sex in a hermaphrodite or pseudohermaphrodite is not easy. The appearance of the external genitals in such a case does not give a correct indication of its gonadal sex. Scientific investigations such as radiological examination of the pelvis and the skull, biochemistry of blood, B.M.R. determination, even hormone estimations have not been of much use. Laparotomy with section from the gonads and biopsy appeared to be the only sure way of diagnosing such a case.

But in the light of recent work we know that in a pseudohermaphrodite the genetic sex may be different from its gonadal sex. A male pseudohermaphrodite is usually a genetic female. Such individuals usually have feminine instincts and emotions and sex desires and feminine external genitals. The question naturally arises if such individuals should be classed as males only because they possess testes. Many leading gynæcologists and scientists would class them as females and direct their treatment so that they may remain as such. Naturally psychological studies are finding an important place in the ultimate declaration of sex in a pseudohermaphrodite.

When we come to the question of treatment we face an equally difficult problem. Study of recent literature does not give clear indication as to the best way of dealing with such individuals. Leading gynæcologists sometimes hold entirely different views as to the best way of dealing with a hermaphrodite or a pseudohermaphrodite. Inspite of these uncertainties, the plan that has appealed to most surgeons is to help these individuals in retaining their declared sex. This gives them best chance of mental adjustment. Mental instincts and desires of the individual are given highest consideration.

With such ends in view the enlarged clitoris has been removed, vagina lengthened, and testes removed in male pseudohermaphrodites. But from the recent advances in our knowledge of endocrinology we know that abdominal testes need not be removed and the same surgeons, who previously removed both testes would probably now only transplant them from their subcutaneous positions to the inside of the abdomen.

Case Reports

Case 1—J. D., aged 20 years, unmarried was admitted into the Lake Medical College Hospital complaining of having never menstruated in life.

Family History—Parents were normal. They are both dead. Patient had 3 elder sisters, all married and two had children. Patient had a twin sister who was unmarried and also had not menstruated as yet.

PAST HISTORY—No serious illness.

PRESENT ILLNESS—Patient had always noticed some enlargement of her clitoris. At the age of 12 or 13 years she began to develop axillary and pubic hairs and some enlargement of the breasts. Since then she had noticed some further enlargement of the clitoris. She had never menstruated.

GENERAL EXAMINATION—Appearance feminine. No hair on the face or chest. Some hair on the axilla. Pubic hair in fair quantity with feminine distribution. No laryngeal prominence. Voice feminine. Breasts slightly developed (Fig. 1, *vide* Plate).

Height—5 ft., Weight 100 lbs.

No abnormality detected in heart or lungs.

INGUINAL REGION—In the left inguinal region there was an oval enlargement 1½"×1" in size, solid, slightly tender. The swelling could not be reduced into the abdomen, but could be pushed down into the left labium majus. No impulse on coughing. No abnormality detected in the right inguinal region.

EXTERNAL GENITALS—The clitoris was enlarged and looked like a penis (Fig. 2, *vide* Plate). It was 2" in length and had a well developed glans and a prepuce. There was a shallow groove on the under surface of the phallus.

Labia majora were well developed with some pigmentation and fine wrinkling of the skin. No gonad in the labial swellings. Labia minora well developed. Vestibule was normally developed and had urethral and vaginal openings, in the usual feminine positions. The vaginal opening admitted the index finger and its depth was 1¼". It was a blind pouch with no trace of the cervix.

P.R.—No uterus or ovary could be palpated.

Blood count, blood chemistry, and urine showed no abnormality.

Skiagram of the pelvis showed a female type pelvis.

Larynx was examined. Vocal cords were rather larger and the cavum of the larynx bigger than what was normal for a girl of that age.

B.M.R. was + 11%.

Mental outlook and instincts were feminine.

OPERATION—(1) Laparotomy was done. Uterus, fallopian tubes, and ovaries were all absent. A gonad was present over the right psoas muscle. On macroscopic examination it appeared to be a testis 1"×¾"×½". Section was taken for biopsy.

(2) The gonad from the left inguinal canal was removed. Macroscopically it also appeared like testis 1¼"×¾"×¾" with well developed gubernaculum and epididymis.

(3) The enlarged clitoris was removed.

HISTOLOGICAL EXAMINATION OF THE GONADS—Both gonads revealed typical structure of testis with well developed glandular elements but no evidence of spermatogenesis. The section from the abdominal gonad showed some degenerative changes.

Result—Recovery from the operation was uneventful and the external appearance of the vulva quite feminine (Fig. 3, *vide* Plate). The patient appeared to be happy.

B.M.R. repeated on 5-9-50 and 2-10-50. On both occasions it was—12%.

Comment—This was a case of male pseudohermaphrodite with feminine appearance and feminine instincts and brought up as a female. Decision was made to help her to retain her feminine status. The enlarged clitoris was removed to relieve some of her mental embarrassments. The left gonad was removed as it was exposed to trauma, and as it could not be transplanted easily into the abdomen. The right gonad was left behind, as that was likely to produce less androgens. The B.M.R. results were interesting as they were definitely of the female type after the removal of the left testis and inspite of the right testis being left behind in the abdomen.

On further enquiries it was revealed that the other twin sister was exactly like this one with penis-like enlargement of the clitoris. It is a pity that we could not get her into the hospital inspite of our best efforts.

Case 2—Miss S. reared up as a female aged 10 years admitted into the Hospital on 20-11-50.

The relatives said that there was some abnormality in the external genitals since the time of her birth.

FAMILY HISTORY—Both parents were alive. Patient had 2 brothers and 3 sisters who were all normal.

PAST HISTORY—No serious illness.

GENERAL EXAMINATION—Well developed and fairly nourished girl. Trunk and limbs quite proportionate. Appearance and behaviour feminine. No evidence of any endocrinal dysfunction. No abnormality detected in the other organs. Breasts and axillary hairs not developed.

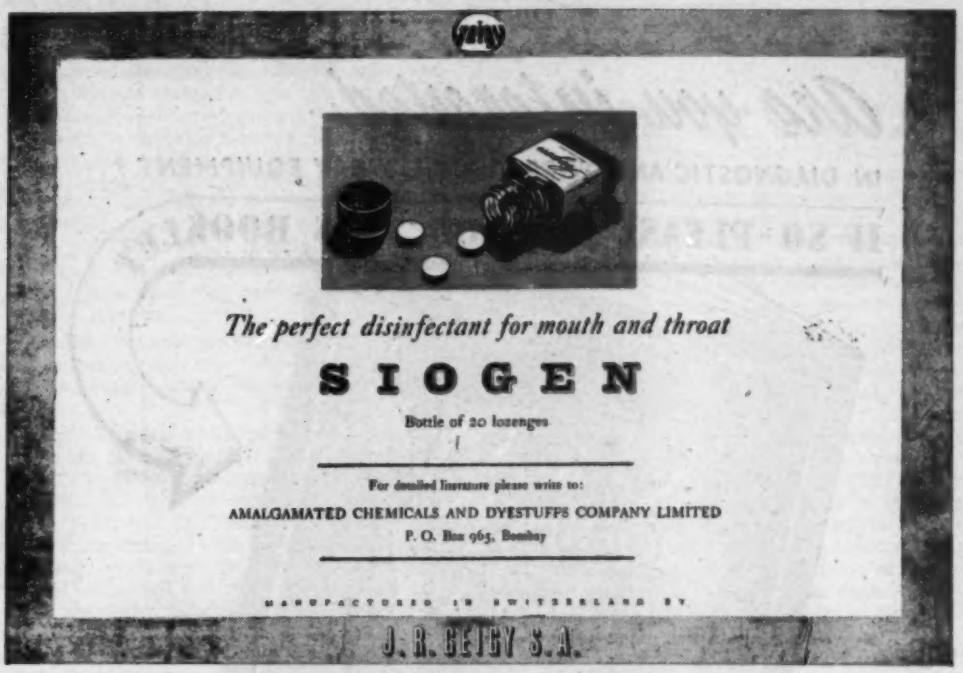
Inguinal canals—No hernia, no swelling.

EXTERNAL GENITALS—The clitoris was enlarged, it was 1½" in length and about ½" in diameter with well-developed foreskin and glans (Fig. 4, *vide* Plate). Near the base of the clitoris was a semilunar opening which was thought to be the urethral orifice. Mons pubis and labia majora had scarce pubic hairs. The skin of the labia majora showed fine wrinlings and some pigmentation. The labia minora and the vestibule were absent.

On passing a catheter it passed into a pouch but no urine was obtained. But immediately afterwards the patient passed urine through the same opening. This led us to suspect the presence of 2 pouches communicating with the external opening, one being the bladder and the other the vagina.

For confirmation skiagraphy was done. The bladder being visualised by descending cystography (after I. V. Iodexyl) and the vagina begin visualised with 10 per cent sodium iodide injected through a catheter passed through the perineal opening. Skiagrams demonstrated the presence of two pouches.

P.R.—Small uterus could be palpated.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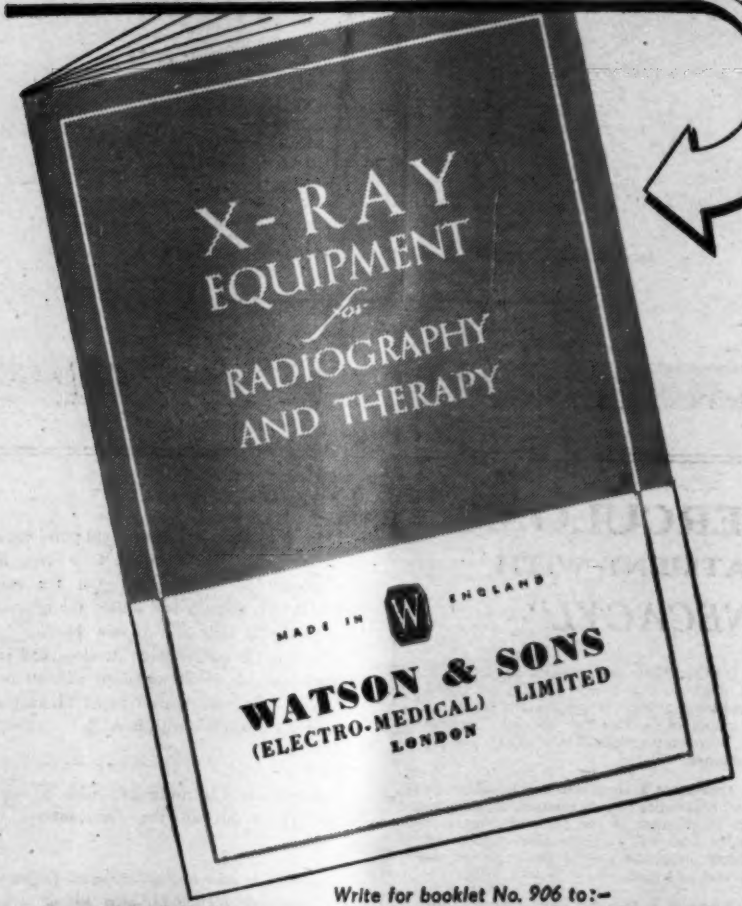

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B.M.R. was —15%.

TREATMENT—Exploratory laparotomy was done on 6-12-50 for further study of the pelvic organs. There were normal sized uterus, fallopian tubes and ovaries. Both ovaries showed a few follicles on the surface. A piece of tissue was removed from the right ovary for biopsy. Left ovary was not disturbed as it appeared to be exactly alike. At the same sitting, perineum was incised in the midline for about 1½" over a catheter passed through the perineal opening. This exposed the vestibular cleft with the urethral and vaginal openings. The vaginal opening admitted the tip of little finger. The labia minora were reconstructed by stitching the mucous membrane of the vestibular cleft to the skin margins.

After 3 weeks the enlarged clitoris was amputated and plastic operation was done for reconstruction of the vulva. The vaginal openig was enlarged. (AG 5, *vide* plate).

COMMENT—This was a case of female pseudohermaphrodite with feminine appearance and feminine instincts, normally developed female gondas and female internal genitalia, but with somewhat masculine external genitals. There was no difficulty in coming to the conclusion that the enlarged clitoris had to be removed and that a plastic operation on the vulva had to be done. It was at first considered that these operations should be done after the patient attains puberty. But from a consideration of the continued psychological effects of these abnormalities in the external genitals, it was decided that the plastic operation on the vulva should be done now, so that the patient could grow and develop with a more balanced mental condition.

SUMMARY

1. The terms hermaphroditism and pseudohermaphroditism are defined and their frequency discussed.

2. Modern conception about the etiology of pseudohermaphroditism or intersexuality is summarised and discussed.

3. Problems in the diagnosis of sex in a pseudohermaphrodite and in the management of such a case are discussed.

4. Two cases of pseudohermaphroditism are reported.

ACKNOWLEDGMENT

I wish to express my thanks to Dr. R. Nag M.B., for taking the photographs and to the department of biochemistry, Lake Medical College, Calcutta for determining the B.M.R. of these two cases.

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PRACTITIONERS' CORNER

CONVULSIONS IN INFANCY AND CHILDHOOD

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Convulsive disorder which is common in pediatric practice is known for centuries, but its ultimate cause is not yet known. Recent studies which include careful clinical examination, the physico-chemical and electro-physical aids bring the physicians nearer to the cause and make the treatment more logical. Clarke in the 19th century described convulsions associated with carpopedal spasm and laryngismus. After him Huglings Jackson and Soltman made their contributions by the end of the 19th century. According to Soltman, the central nervous system in children is immature and therefore hypersensitive to peripheral stimuli. Some writers of the 20th century have grouped all convulsions in one fundamental unit, whatever may be their causes (Collier, and Kennedy). Others classify these convulsions into two groups; (1) epileptic and (2) symptomatic. The latter includes convulsions due to tetany, uræmia and intracranial diseases. In Germany, some described a 3rd group considered to be benign recurrent convulsion mostly occurring at the onset of illness in infants and children. In the present century writers have tried to show the relationship of infantile convulsions to epilepsy. Epilepsy is regarded as a chronic convulsive disorder with sequela of progressive mental deterioration. It is observed by some that only 12 per cent infantile convulsions are followed by recurrence and probable mental degeneration. If there is no history of epilepsy in the family and the child had no convulsions before except one at the onset of an acute infection, this single convulsion is regarded as innocent. Mental degeneration is seen when there is disturbance of health or the child has many fits. The study of the tracings of the electroencephalograph in epileptics and normal persons throws more light on this problem. This tracing detects potential epileptics. The electroencephalograph alone will not be confirmatory as in a few cases of those getting convulsions, the tracing may be normal. Relatives of epileptics, even though they may have no convulsions show abnormal tracings, (potential epileptics).

Convulsions in childhood may or may not be associated with diseases. Acute respiratory tract infection forms a large group, primary diseases of the central nervous system, less than half of the total cases and epilepsy forms a small group. Hypocalcæmia was considered the main cause of convulsions in children under three years until the beginning of the present decade. But to-day because of preventive measures *viz.*, adequate administration of vitamin D and calcium, the incidence of convulsions is much less. At the onset of fever, there is temporary alkalæmia which depresses ionization of calcium and makes neuromusculature more excitable and convulsions are precipitated. This is more usual in children with latent tetany. Theory of innate proclivity is postulated by some workers. All children with tetany do not get convulsions even in the presence of acute infection such

as meningitis, encephalitis etc. This shows that hypocalcæmia is not the real cause of convulsion but only a precipitating factor. The inherent convulsive tendency also varies in degree. When it is highest convulsions are precipitated without any obvious cause—idiopathic epilepsy. In another group the innate tendency is insufficient and only a provocation by a severe stimulus will produce the fit, such as in acute inflammation of the meninges. According to Peterman, the incidence of convulsions in infancy and childhood is high because convulsive threshold in children is slightly lower. The view about hyperirritability of infantile nervous system is not accepted because only a few children have convulsions while they are young. For clinical purposes and from the prognostic point of view they are typed as:

(1) Spontaneous which include idiopathic epilepsy; Convulsions with mental defect, developmental abnormality of the brain and convulsions resulting from permanent brain damage.

(2) Provoked convulsions which include acute inflammation of meninges, tetany, uræmia and the initial convulsions of acute infection. Furthermore in certain borderline cases provoked convulsions will cause sufficient damage to the brain to tip the scale in favour of spontaneous convulsions thereafter.

The word epilepsy can be used in place of convulsions as all convulsions are grouped in one fundamental unit. But to avoid confusion, the word epilepsy is reserved for the well-known clinical picture and the convulsion is retained in the classification carrying a wider meaning and including other related seizures.

Classification

The different types are classified in 3 ways: (*i*) Clinico-anatomically, (*ii*) Ætiologically and (*iii*) Chronologically.

Etiologic classification of convulsive disorders (Mcquarries' classification modified by Peterman 1947):

1. *Acute or non-recurrent convulsions*:—
   - (*a*) Extracranial infection.
   - (*b*) Intracranial infection.
   - (*c*) Intracranial hæmorrhage.
   - (*d*) Toxæmia.
   - (*e*) Anoxia.
   - (*f*) Metabolic disturbances.
   - (*g*) Acute cerebral œdema.
   - (*h*) Tumors of the brain.
2. *Chronic or recurrent convulsions*:—
   - (*a*) Epilepsy.
   - (*b*) Pyknolepsy.
   - (*c*) Narcolepsy or cataplexy.
   - (*d*) Hysteria.
   - (*e*) Tetany.
   - (*f*) Hypoglycæmic states.
   - (*g*) Uræmia.
   - (*h*) Cerebral allergy.
   - (*i*) Cardiovascular dysfunction.
   - (*j*) Parasitic diseases of the brain.
   - (*k*) Intracranial neoplasms.
   - (*l*) Migraine.

Peterman from an analysis of 2500 cases with convulsions mentions the following as causative factors: 33·4 per cent acute infection, 26·3 per cent idiopathic epilepsy, 14·2 per cent cerebral birth injury or residue; 13·1 per cent miscellaneous causes; 7·4 per cent infantile tetany; 5·6 per cent of unknown cause.

Chronological classification by Peterman is as given below.

For a comparative study cases of convulsions from B. G. Hospital, Bombay (record of 1947) are included in the tables:—

A. Birth to 1 month

Diseases	*Peterman's record No. of cases (7 per cent of the total)*	*B. J. Hospital's record No. of cases*
Cerebral birth injury ..	122	1
Acute infection	18	2
Hydrocephalus	6	2
Idiopathic epilepsy ..	6	nil
Infantile tetany	5	4
Meningitis	3	2
Uraemia	1	nil
Cerebral agenesis	1	nil
Congenital heart disease ..	1	nil
Congenital syphilis ..	1	nil
Unknown	12	nil

B. 1 month to 6 months

Diseases	*Peterman's record No. of cases (12·9 per cent of the total)*	*B. J. Hospital's record No. of cases*
Acute infection	102	3
Infantile tetany	61	15
Cerebral birth injury & residue	59	nil
Cerebral agenesis	18	nil
Idiopathic epilepsy ..	15	nil
Meningitis	10	16
Hydrocephalus	6	3

C. 6 months to 3 years

Diseases	*Peterman's record No. of cases (47·2 per cent of the total)*	*B. J. Hospital's record No. of cases*
Acute infection	554	5
Idiopathic epilepsy ..	197	4
Cerebral birth injury and residue	123	nil
Infantile tetany	120	16
Meningitis	37	23
Acute & chronic encephalitis	26	8

D. 3 years to 10 years

Diseases	*Peterman's record No. of cases (27·4 per cent of the total)*	*B. J. Hospital's record No. of cases*
Epilepsy	332	8
Acute infection	158	6
Cerebral birth injury ..	48	nil
Acute & chronic encephalitis	33	5
Meningitis	12	8

E. 10 YEARS TO 16 YEARS		
Diseases	*Peterman's record No. of cases (5·9 per cent of the total)*	*B. J. Hospital's record No. of cases*
Idiopathic epilepsy	107	2
Acute & chronic encephalitis	8	2
Congenital syphilis ..	5	nil
Cerebral birth injury ..	4	nil

The above tabular analysis and the findings of Perry Cramp, show that the incidence of convulsions caused by acute infection is higher in the younger age groups and the incidence of chronic convulsive disorders is higher in the old age groups. From birth to 16 years, in the first six months the incidence is lowest and in the period from 6 months to 3 years it is the highest. Sexes are equal. In patients with encephalitis less than 30 per cent had convulsions in B. J. Hospital series and with tetany 95 per cent had the seizures. The incidence of organic epilepsy is greater in the younger age groups, while idiopathic type in the older age group. In ascaris lumbricoides infestations, the toxin "ascaron" secreted by the worms precipitates in severe cases symptoms of delirium, meningism and convulsions. This is common in young age. In febrile convulsions, the convulsions occur when there is a sudden rise of temperature. Commonest age is 2 years. The following infections precipitate convulsions more commonly than others—

(1) Upper and lower respiratory tract infections due to pneumococcus, haemophilus influenza bacillus and other organisms.

(2) Middle ear infections.

(3) Whooping cough.

(4) Uræmia associated with fever specially when temperature rises.

(5) Measles, influenza, chicken pox.

(6) Pyelitis, poliomyelitis, mumps and encephalitis.

Diagnosis

In adults and also in later childhood, there is usually a definite history of occurrence of fits in phases—a sensory aura, a cry followed by loss of consciousness, a tonic phase and a clonic phase of motor activity, recovery of consciousness and subsequent sleep. In infancy we do not get such a picture. A patient is usually brought or the message is given by his relatives, that the child is getting fits or convulsions. A sudden twitching of a voluntary muscle, a group of muscles or a number of a group of muscles with partial or complete loss of consciousness is called a fit or convulsion. The spontaneous jerk of the finger or a limb of a normal person is presumably the result of a sudden involuntary and uncontrolled discharge of stimuli from a group of neurons in the motor cortex and is different only in degree from convulsions. An infant's irregular, restless, muscular movements are of the same quality. As growth progresses these irregular jerks disappear, to be replaced by smooth voluntary movements. In major convulsions, the child or adult suddenly and temporarily returns to the state of irregular jerky movements of an infant. The history from parents many a time helps in diagnosing the case. But at times the slight transitory or localised symptoms may be entirely overlooked and the diagnosis becomes very difficult. Sometimes the child is brought with the history of "blue attacks", "fainting fits" and these are attributed to teething or wind. These are usually accompanied by momentary loss of consciousness with a fixed stare, convulsive twitchings of short duration or clonic eye movements. When a child is brought for convulsive attacks, it must first be found out whether the exciting cause was of an intensity to produce the convulsion in a normal child or whether there is pathological instability of the central nervous system. Also the predisposing factors like malnutrition, debility, or heredity in producing the pathological instability, must be found out. All the etiological factors which are tabulated above must be kept in mind according to the age of the child. Examination of blood for total and differential counts, malarial parasites, estimation of calcium, phosphorus and phosphatase, glucose etc., helps in diagnosis. Complete examination of cerebrospinal fluid, urine and stool, x-ray examination of the skull and the epiphysial ends of the long bones, and of the chest will give an idea of the associated diseases. Complete physical examination is very important. The difference between a "choking fit" and a fit which causes "choking" must be remembered. Laryngeal spasm in whooping cough or a functional spasm may precipitate a fit. Laryngeal spasm in tetany is known as laryngismus stridulus. In short, age, birth history, family history, showing the constitutional instability of the nervous system with reflex irritability arising from common cold, earache, worms, and gastrointestinal disorders, the presence of fever, (site of infection, infectious disease and epidemicity), focal signs pointing to brain lesion, (localised paralysis, rigidity, changes in size and reaction of the pupil and strabismus), recurrent frequent convulsions (due to intracranial hæmorrhage or meningitis), lead line on the gums, history of birth, (premature, miscarriages, still births) and signs of specific diseases, all these points are diagnostic aids.

Prognosis depends upon the cause; but the mortality is far less to-day because of the recent advances in investigations and in therapeutics which have brought the physicians nearer to the cause and made the treatment more logical.

Treatment

The proper treatment of a convulsion must begin with attempt to determine the cause. Treatment essentially comprises of—

(1) Treatment of the emergency and (2) treatment of the cause.

Lennox describes the therapy of convulsions and epilepsy in the following words, "Fit the treatment to the fit." He used the drugs, selection of which he made for stabilization of specific cortical dysrhythmias and for specific seizures. He describes four seizure patterns: (1) convulsive seizures, (2) psychomotor seizures, (3) Petit mal, and (4) diencephalic seizures. He fits his treatment according to the seizure pattern. For group 1—in order of preference—phenobarbitone, dilantin, mesantoin, mebaral and bromide. For group 2—dilantin alone. If it is not effective tridione or mesantoin may be added. For group 3—tridione is the drug of choice. This group is resistant to drug therapy. For group 4—amphetamine sulphate or one of the anticonvulsant drugs is chosen. At times, the same patient may have different patterns mixed. In such cases, patient therapeutic testing will give the proper therapy.

For successful therapy and prevention of recurrences certain points should be considered.

USE OF THE FOLLOWING DRUGS: Phenobarbitone sodium by subcutaneous injection. 1-2 grains is effective. This should be repeated, if necessary. Effect is seen within 15-30 minutes. The doses according to age are as follows:—

Infants under 1 year	..	¼ gr.
Children between 1-5 years	..	¼—½ gr.
" " 5-10 "	..	½—1 gr.
" " 10-15 "	..	¾—1½ gr.

This should be repeated 4-hourly till the convulsions disappear for 24 hours. Intramuscular injection of paraldehyde 1-2 ccs. is much more effective and is without danger. Dr. G. Coelho of Bombay advises occasionally drawing out of C.S.F. by lumbar puncture and administration of chloral hydrate, pot. bromide mixture orally or sodium seconal capsules per rectum. This makes the child fall fast asleep within 10-15 minutes and the brain gets the needed rest. Further trauma is thus prevented.

Minor convulsions are eliminated by tridione. When combined with phenobarbitone it is the most useful prophylactic treatment against minor convulsions. Failures in controlling convulsions is due to laxity in regular and accurate medication, than to inefficiency of medical treatment itself. Chloral hydrate 3-15 grains 2-4 hourly per rectum, depending upon the age of the patient controls the convulsions when oral administration is not possible.

THE AVOIDANCE OF THE FOLLOWING: (1) Injury to the patient, (2) opiates, (3) morphine or codeine, because, they act on higher centres, depress the respiratory centre and inhibit peristalsis, (4) ether, because excitement stage is too long and stimulating, (5) hot baths, as this will raise the temperature and may cause burn. Douglas Buchanan of Chicago is of the opinion that enemas and hot and cold baths during a convulsion serves less to control convulsions than to satisfy and occupy the minds of parents who are afraid of the child's condition.

If the temperature is high, measures for its reduction (sponging, wet packs, cold water enema, etc.) are adopted. Complete rest is advisable. For convulsions in acute nephritis, 50 per cent magnesium sulphate ½-1 oz. 4-hourly per rectum or 2-8 c.c.s by intramuscular injections 4-hourly or 25 per cent mag. sulph. 2-5 ccs. slowly intravenously.

TREATMENT OF SPECIAL TYPE OF CONVULSIONS: *Acute infectious diseases*—as sudden rise in the temperature precipitates the attack the treatment is to adopt measures to reduce the temperature. Specific treatment is given if indicated. Enema with cold water, sponging with cold or tepid water or wet packs, do help in lowering the temperature. Acetyl salicylic acid is given three times a day. Focus of infection is found out and treated accordingly.

Tumour of the brain—uncommon; treatment is surgical.

Congenital cerebral defect—no treatment for defect; anticonvulsant drugs are used to check convulsions.

Congenital syphilis—treatment of convulsions first and then antisyphilitic treatment.

Direct trauma—general measures of convulsions when no external evidence of haemorrhage, fracture or laceration is evident.

Gastroenteritis—in this condition, convulsions are precipitated by toxæmia and/or the fever.

Hydrocephalus—saturated solution of magnesium sulphate orally and when there is intracranial tension, detected by increased tension of the anterior fontanelle, 25-50 per cent magnesium sulphate, 1-2 cc. intramuscularly daily is useful. Lumbar puncture or ventricular or cisternal puncture for the withdrawal of fluid will be of much help. After withdrawal of the fluid, head of the child is kept on lower level and not on a raised surface. Amount of fluid to be withdrawn depends on the intracranial tension. When possible, the underlying cause can be removed by surgical measures.

Intracranial abscess—surgical treatment and treatment of the convulsions instituted.

Intracranial hæmorrhage—It may be focal or generalised. Chloral hydrate orally or per rectum—1 to 3 grains or sodium phenobarbitone ¼—½ grain give rest to the patient and prevents further injury, concentrated solution of magnesium sulphate may be tried intramuscularly. If there are symptoms of tension, lumbar puncture should be performed cautiously as this procedure may increase the hæmorrhage, because of decreased intracranial pressure. Surgical measures should be adopted if there is rupture of middle meningeal artery.

Meningitis—general measures as usual and active specific treatment of meningitis.

Strychnine poisoning—Usually there is a history of taking a tonic containing strychnine. Intravenous in-

jection of pentothal sodium is the treatment of choice. Tracheal insertion of a catheter to overcome laryngeal spasm and then oxygen administration by tent or mask gives relief. Sodium phenobarbitone 1½—3 grains per rectum is administered till the patients get no convulsions for 12-24 hours.

Tetanus—stimulus of any kind should be minimised as this will precipitate more convulsions. Patient should be kept in a darkened room and handled as little as possible. He should be given nasal feeds and intravenous administration of fluids, such as saline by drip method, anticonvulsants like sodium phenobarbital or sodium amytal should be given intravenously. Intocostrin ½—¾ gr. intravenously or intramuscularly, 3-hourly should be used for relief of pain and spasm. Intravenous injection of antitoxin in a large dose, 50,000 units, with 500 ccs. of physiological saline is administered slowly at the rate of 60 drops a minute. If patient gets rigors, the remaining amount should be given intramuscularly. Before injection is given, test for sensitivity is performed.

Tetany—This is usually seen upto 3 years. Calcium chloride is the drug of choice. It contains 30 per cent calcium while calcium gluconate contains 9 per cent calcium and calcium lactate contains 10 per cent. Calcium gluconate 10 c.c. intravenously or intramuscularly 2·5—5 c.c. 12-24 hourly or at frequent intervals if the condition is more acute. Calcium chloride initially 2 grams per day and after 2-3 days, the amount is reduced to avoid the development of acidosis. In premature baby the dose is lesser than that for a normal child. In acute cases and thereafter till the convulsions are stopped for 24 hours chloral hydrate is added to calcium chloride mixture. Chloral hydrate can be given orally or per rectum. The dose of chloral hydrate according to age is as follows:

0-6 months	3-5 grains
above 6 months	5-10 „

Calcium chloride-chloral hydrate mixture, *viz.*,

Calcium chloride	30 grains
Chloral hydrate	16 „
Potassium bromide ..	16 „
Tincture card. co.	20 minims
Syrup caldion	2 drachm
Aqua ad	1 ounce

Mft. mixture: the dose is according to the age and acuteness of the condition and is to be given 3-4 hourly.

Vitamin D should be administered for a long time. Dihydrotachysterol 2·5 mgm. a day orally in hypoparathyroid conditions is preferred to parathyroid extract which has the following disadvantages: (1) action is abrupt, (2) action is short lived and (3) has toxic effect.

Uræmia—This in the course of nephritis may precipitate convulsions. Treatment is the same as for convulsions in general. Lumbar puncture is performed to reduce the cerebral œdema. Intravenous injection of 2 per cent magnesium sulphate should be given at the rate of 2 c.c. per minute till blood pressure comes to normal level. Later on saturated solution of magnesium sulphate should be given orally till blood pressure remains stable. Large amount of fluid is administered.

Epilepsy—This condition is very difficult to treat as the cause cannot be accurately found out and therefore a symptomatic treatment is only possible. Mental factor is very important in precipitating the attack. Nothing about this condition should be discussed in the presence of the patient. A tactful social worker or a school teacher who is intimately in contact with the family can give valuable service in this direction. If the attack can be checked by drugs, a child can be allowed to attend school for normal children. His education and all activities should be allowed to run on the same lines as those of normal children. But if the attacks are severe and frequent these children should be sent to the school for epileptic children or to institutions for epileptics.

It is generally agreed that an epileptiform attack is due to an "explosion" of abnormal impulses in some portion of the brain, causing disorderly responses in the neuromuscular mechanism, frequently accompanied by an interruption of conscious processes in the cerebrum. The main basis of treatment is to reduce this abnormal irritability and thus reduce explosion. Sedatives and anticonvulsants but not soporific drugs should be given. The object of treatment is the complete prevention of seizures with as little interference as possible with mental, emotional development and physical activity. Bromide was the first drug of choice. Next is phenobarbitone. Third one is mebaral, closely related to phenobarbitone. In 1938, Merriet and Putnam introduced dilantin sodium. All these are useful in *grand mal* or generalised convulsions. For *petit mal* tridione is given. This drug may depress bone marrow and cause agranulocytosis and may increase or precipitate potential grand mal seizures. So in case of doubt or in mixed types of convulsions, tridione should be given with any one of the above anticonvulsant drugs (phenobarbitone, dilantin etc.). Dose of anticonvulsant drugs should be increased gradually till attacks cease. This treatment is continued for about five years and then gradually discontinued.

Ketogenic diet is most useful in checking the epileptic attacks. This consists of ketogenic and anti-ketogenic diet in the proportion of 3:1 (Woodyatt's formula).

This diet should be started gradually to avoid neusea and vomiting. The carbohydrates are then decreased with an increase in the fat in 4-5 days' time. This diet should be continued for 6-12 months. Then the carbohydrates are increased gradually with reduction of the fats in the diet till normal diet is given. Dietetic treatment is found to be more effective than drug therapy. When both drug and dietetic treatments are given, the drug should be continued for some more time even when dietetic change to normal is made. Along with this dietetic treatment, vitamin supplement should not be forgotten. Intake of water per day is also gradually restricted.

TREATMENT SERIES

TREATMENT OF CEREBRAL VASCULAR ACCIDENTS

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Calcutta

Next to meningitis and functional nervous disorders like psychoneurosis, hysteria and epilepsy, the cerebral vascular disorders are the commonest neurological conditions which a general physician may be called upon to treat. These disorders usually present themselves as emergency medical problems and are rightly labelled 'vascular catastrophes' or 'vascular accidents' from the seriousness of their nature. The 'apoplexy' of the laity includes all these vascular catastrophes. Of these, cerebral thrombosis is the most frequent. Hypertensive encephalopathy is nearly as common as cerebral thrombosis, at least in private practice. Embolism is distinctly rare but intracerebral or subarachnoid hæmorrhage is fairly common.

In the management of these disorders certain objectives of treatment should be clearly understood. In the first place, an attempt should be made, as promptly as possible, to diagnose the nature of the lesion. One should then proceed to check, eradicate or reduce the severity of the essential pathological process of the catastrophe, that is, the spasm, the thrombosis, the hæmorrhage or the embolic process. It is necessary to admit here that the diagnosis of the exact nature of vascular lesions may not be possible in quite a large number of the cases, even after application of the known canons of differential diagnosis. It is probably a wise dictum that if one cannot distinguish satisfactorily between thrombosis and hæmorrhage one should treat the case as thrombosis.

Even when we are able to diagnose the nature of the lesion, it has to be remembered that barring relief of vascular spasm and a questionable retardation of the thrombotic process, probably we have no effective means of combating the primary pathological process. Fortunately in many non-hæmorrhagic cases, the process halts, reverses or lessens in intensity by spontaneous mechanisms and it cannot be always asserted that a particular therapeutic procedure is responsible for the favourable turn. In hæmorrhage cases, whether with any treatment or without it, such favourable end results are quite rare but not impossible.

The second objective in treatment is based on a consideration of the secondary pathological changes in the brain following upon the initial vascular accident. After a thrombo-embolic or spastic occlusion of a cerebral vessel, there is a stasis and increased permeability of the capillaries ahead; loosening of the brain tissue with loss of support to the walls of the capillaries and arterioles; a diapedesis of red blood cells and white blood cells and transudation of fluid; and even a necrosis of the vascular walls and extravasation of blood in the affected area of the brain. The immediate result of such process is œdema of the brain tissue with or without an underlying ischæmic brain softening. The process of œdema spreads to adjacent regions sometimes at a dangerous pace, sometimes slowly, taking a few weeks' time and sometimes after false lucid intervals, involving the vital regions of the nervous system. Also it has been realised, that in some cases, there may be a vasospasm in widespread regions of the brain following reflexly upon the initial vascular accident, thus causing a widespread disturbance of cerebral circulation. The spreading œdema and, in some cases, the reflex widespread vasospasm are of extreme importance as these intensify the cerebral dysfunctions and precipitate or prolong the comatose state. In a given case the existing cerebral œdema may be worsened by asphyxia, expiratory strain, respiratory diseases like emphysema and pneumonias and cardiac failure.

The third objective in the treatment of cerebral vascular accidents is to prevent, anticipate and treat promptly cardiorespiratory complications (cardiac failure, pulmonary œdema, pneumonias) which tend to occur as terminal events, adding insult to injury.

Finally, if the patient survives the acute attack, attempts should be made to rehabilitate the disabled person with appropriate measures and means should be devised to prevent or defer fresh catastrophes.

Let us now examine the various therapeutic steps which can be taken to attain the various objectives outlined above.

1. To check, minimise or eradicate the immediate pathological process responsible for the accident.

In most instances, hæmorrhage cannot be stopped by any therapeutic procedure. The use of coagulants by appropriate methods of administration is of doubtful value unless there are specific deficiencies which account for rare instances of cerebral hæmorrhage *viz.*, vitamin C and vitamin K deficiencies. Parenteral use of calcium preparations, vitamins C and K, and coagulants like coagulen, wholeblood and congo-red is not followed by any impressive result. Whether venesections and withdrawal of a few ounces of blood would materially contribute to cessation of intracerebral and subarachnoid bleeding has yet to be proven. For practical purposes, however, one may employ venesection and catharsis within 24 hours of the attack, if blood pressure is very high, particularly the systolic (190 mm. Hg. or more). In cerebral hæmorrhage the height of the systolic pressure and of the pulse pressure rather than that of the diastolic pressure is of considerable significance. This has also been pointed out recently by Gupta (1950).

Theoretically, one would wish to employ surgical measures to ligate the bleeding vessel but in practice this is not feasible and has not yet been done. However there are a few records of successful life saving surgery securing evacuation of the blood clot from the brain (Penfield 1933).

Cerebral thrombosis has been treated with heparin and dicoumarol in some instances, with a view to

retard the intravascular clotting. Occasional success has been claimed but the present writer has yet to see a case definitely benefited by such therapy. Probably the employment of these anticoagulants yields more success in the treatment of cerebral venous thrombosis (e.g. in puerperal cases) (Hyland 1950) than in the usual case of arterial thrombosis. If an antecedent fall of blood pressure or dehydrating process has been contributory to thrombotic process, circulatory stimulants and fluid therapy should be judiciously employed. In syphilitic cases, appropriate antisyphilitic measures should be instituted, particularly with a view to clear the associated inflammatory lesion.

In embolism, the treatment is the same as in thrombosis. In cases of angiospasm, as is met with in hypertensive encephalopathy, cerebral vasodilators should be employed, *e.g.*, nitrites, nitroglycerin, aminophylin, etc. Regarding aminophylin, opinion is divided as to its action on the cerebral blood vessels. Cerebrospinal fluid pressure is known to diminish after injection of aminophylin (Kety and Wechsler, 1950), but whether it is cerebral vasodilator (Bouekaert and Jourdan, 1937; Schmidt, 1944) or a vasoconstrictor (Kety and Wechsler, 1950) requires further elucidation. Clinical experiences of the present writer suggest that aminophylin is useful in relaxing cerebral vascular spasm if that be the pathology of hypertensive encephalopathy. Aminophylin may be helpful in these cases in other ways too: by opening out the renal and coronary circulations or by lowering the systolic pressure. Papaverine has also been used for relaxing the cerebral blood vessels and some have obtained the same result by using nicotinic acid. Incidentally, the above drugs are useful in relaxing the secondary vasospasm which follows reflexly in some cases upon an initial accident.

Recently stellate ganglion block with procaine solution has been tried (Gilbert and de Takats, 1948; Naffziger and Adams, 1950; Amyes and Perry, 1950) with a view to dilate the cerebral blood vessels and results have been claimed to be satisfactory, although from a physiological point of view the sympathetic nerve supply of the cerebral blood vessels is feebly functioning. In view of the findings of Kety and Harmel (1948) and others like Scheinberger (1950) that cerebral blood flow does not increase after stellate ganglion block; the role of this procedure in the treatment of cerebral thromboembolic or spastic lesions remains *sub judice*.

2. To check, localise and minimise the secondary cerebral œdema.

Various measures may be adopted for the above purpose, like intravenous injection of concentrated solutions of glucose, sucrose or human albumen; rectal administration of concentrated magnesium sulphate solution and saline catharsis.

The writer employs glucose solution 50 per cent 50 c.c. intravenous, every 6 to 8 hrs. and has got a favorable impression of its value. Sucrose solution is said to be more efficacious in this respect but some observers have repeatedly demonstrated renal tubular damage following the use of the same.

A propped up position of the patient may lower the cerebral venous pressure which may also be achieved by venesection, particularly in cases where there is a clinical suspicion of increased cerebral venous pressure as in emphysema, bronchial spasm, pneumonias and cardiac failure complicating the cerebral vascular lesion. Lumbar puncture may also be helpful if intracranial pressure is raised in association with cerebral œdema or if the condition is one of spontaneous subarachnoid hæmorrhage. However, it should be done cautiously to avoid the 'medullary cone' phenomenon, particularly in cases where intracranial pressure is markedly raised.

Where œdema threatens the medullary centres, respiratory and vasomotor stimulants like lobeline, coramine, cardiazol and caffeine may be used to prolong life while measures are being undertaken for lessening the œdema.

3. To prevent, anticipate and combat cardiopulmonary complications.

Attention to posture to prevent hypostatic congestion, careful feeding to avoid aspiration pneumonia and a vigilant watch on the cardiac condition may go a long way in preventing these complications or in rendering them obvious early, when they are susceptible to treatment. When these complications do arise, the question of using penicillin, oxygen therapy and digitalis should be judiciously considered.

4. General care.

Good nursing, general care of the skin, bladder and bowels, good clearing of the throat and attention to nutrition and hydration of the patient, supplementing these by parenteral methods where necessary, are as important considerations in the management of the cerebral vascular patient as in the treatment of any other comatose state. For the restless patient, it is better to avoid morphine and barbiturates which may have depressant effects on the respiratory centres. Paraldehyde—rectal or parenteral—serves our purpose very well in these cases.

5. To adopt measures to rehabilitate the disabled person.

This part of the regime is the most neglected one in private or hospital practice. Patients are left to themselves, at the mercy of nature. This negligence amounting to criminal callousness on the part of the attending physician cannot be too strongly deprecated. There is no doubt much can be achieved by sympathy, sincerity, and perseverance. A full range passive movement of the joints of the affected limbs, gentle massage of muscles, re-education and training in speech and movements of the limbs including locomotion, at first aided and then unaided, are measures worth adopting and in a large number of cases these are well-rewarded. From an invalid state, the patient may emerge out as a self-dependent person capable of looking after himself and in some cases may be fit enough to take up light jobs. Instead of being lifelong economic burdens on the family the person may contribute materially to the economic welfare of the same. Usually, what recovery

occurs is manifest from one to three months of the thrombo-embolic process but even those cases which have been neglected for several months may make impressive recoveries under painstaking physiotherapeutic measures.

Prostigmine and phenobarbital therapy have been claimed (Doane *et al*, 1947) to hasten recovery of muscle power but the present writer's experiences with the same have not been so lucky.

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SPECIAL ARTICLE

THE PROBLEM OF CANCER IN ASSAM WITH SPECIAL REFERENCE TO CANCER LARYNX*

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INCIDENCE

The general belief that cancer is not common in India is incorrect. This has already been repudiated by the Bhore Committee in its report and is corroborated by our findings (noted below) in Assam.

Thompson and Negus mention that cancer of the larynx forms only 2 per cent of all cancers, but a study of the figures of the E.N.T. Department of the Assam Medical College for the last two years (October, 1948 to October, 1950) shows the following:

During the period of from October, 1948 to end of December, 1948 there were 18 cases of malignancy in the mouth and throat region. The distribution of these cases were: larynx—15, tongue—1, pharynx—1 and œsophagus—1.

In 1949, 62 cases of malignancy of the same region came for treatment, the distribution being, larynx—57, tonsil—2, palate—1 and tongue—2.

* Based on a paper read at the Second All-Assam Medical Conference of the Indian Medical Association held at Jorhat on the 26th-27th October, 1950.

It may be interesting to compare these findings to the findings of the other departments of the hospital.

During the period the following four cases of malignancy came to the medical ward : stomach—1, œsophagus—1 and liver—2.

The following is the analysis of incidence of cancer in 1950 (from January to 11th October) :

E. N. T. ward	..	63 cases
Surgical ward	..	22 cases
Medical ward	..	9 cases
Maternity & gynæcology ward	..	12 cases
		106 cases

Analysis of 63 cases in the E. N. T. Ward:—

Larynx—42
Oesophagus—11
Tonsil—4
Tongue—2
Bronchus—1
Palate—1
Secondary, of doubtful origin—1
Papilloma—1 (simple)

Analysis of 22 cases in the Surgical Ward:—

Penis—6
Stomach—4
Breast—3
Rectum—2
Lip—2
Nose—1 (outside of the nose)
Oesophagus—1
Testis—1
Neck—1
Liver—1

Analysis of the cases in the Medical Ward:—

Stomach—4
Secondary—3 (doubtful origin to lung)
Oesophagus—1

All the 12 cases in the maternity and gynæcology ward were cases of cancer of the cervix of the uterus. Therefore total cases of carcinoma in Assam Medical College in 1950 upto the 11th October comes upto 106. Of these the largest were in the larynx (42 cases).

In 1948 and 1949 also there were 15 and 57 cases respectively of laryngeal carcinoma, but other comparable figures are not available; yet there is no doubt that they were also in large proportion. This shows that the incidence of the cancer larynx met in this hospital, is the highest.

Thompson and Negus in their book on diseases of the nose and the throat says—"Malignant tumours of the larynx are fortunately less frequent than cancer in many other regions and constitute about 2 per cent of all cancers."

In the "Recent Advances in Otolaryngology" by Scot Stevenson it is mentioned, "Cancer of the larynx

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is much less frequent than cancer of the stomach or uterus....." but the above analysis shows 42 larynx cases against 8 gastric and 12 uterine cases. Whether the above figures of this hospital are applicable to the whole of India cannot be said with certainty as the number and period is too small but one thing can be said definitely from the last two years' experience that the incidence of cancer of the larynx is not negligible and is not so small as is thought to be, specially in Assam.

Types of Carcinoma

All growths arising from the structures above the false vocal cord, *i.e.*, epiglottis, aryepiglottic fold, pyriform fossa both walls, etc. have been included in the extrinsic group and those from below the false vocal cord and from the true vocal cord, have been included in the intrinsic group, and those below the true vocal cord as subglottic.

It is usually said that intrinsic carcinoma of the larynx is the more common than extrinsic, as mentioned in the Thompson and Negus's text book. But an analysis of the 114 laryngeal cases met with so far shows all to be of the extrinsic type except one which is intrinsic; no subglottic type was met so far. The commonest site have been in the pyriform fossa.

In the "Recent Advances in Otolaryngology" it has been pointed out that V. E. Negus met in 20 years of his practice 93 intrinsic and 94 extrinsic cases—almost an equal number. But here in Assam majority of the cases without doubt were extrinsic. If a margin is given for border line advanced cases where origin may be difficult to determine, even then 75 per cent would be extrinsic. In U.S.A. also extrinsic cancer is said to be commoner as mentioned by Acherman and Regato in their book on cancer. Comparison of some of the figures obtained in U.K., U.S.A. with those obtained here reveals a wide variance in them and probably there is the question of racial and geographical and climatic conditions which caused this variance.

Another aspect also strikes one as important, namely, that authority like Negus met only 187 cases of laryngeal carcinoma in 20 years and Semon only 212 cases in 28 years whereas here in Assam the author came across 114 cases of laryngeal carcinoma in 2 years only.

Age Incidence

An analysis of the age groups of these cases also disproves the common belief that these cases are more common beyond the age of 50 years and rare in young people below 30 years.

Dingly gave the following figures for U.K. from one of his analyses :

Age	No. of cases
31—40 years	5
41—50 „	16
51—60 „	30
61—70 „	13

It is to be noted there were no cases below 30 years. Now here in Assam:

Age	No. of cases
21—30	24
31—40	25
41—50	49
51—60	31
Above 60	12

This shows that there were a large number of cases even below 30 years and the largest group has been between 41 and 50 years. Therefore, the common idea given to the students as cancer being rare in younger people should not be taken as true for our country.

Method of Control

For the control of this disease, the problem has got to be attacked from several angles and broadly these can be classified as follows:—

(1) Education of the public to make them cancer-conscious.

(2) Education of the general practitioners to enable them to diagnose the cases early.

(3) Research and treatment centre where causes of the predilection of cancer larynx would be investigated and adequate treatment by surgery and radiotherapy given.

It will be noted that the results of the treatment are unquestionably influenced by the time interval which elapses between the beginning of the lesion and the diagnosis. So that delay may not occur in diagnosis, the public should be educated to consult doctors promptly, on the first appearance of symptoms giving the doctors a chance to diagnose it early. The doctors should be given adequate basic training to enable them to do so.

As Ackerman and Regato point out, "unfortunately, physicians just leaving medical schools are inadequately prepared for their role in the fight against cancer. The medical students acquire a disproportionate idea of the possibilities of treatment and is usually most impressed by the failure or by the incurability of some forms of cancer. The training in cancer which the G. P. should have must be given him either in his medical school or through channels easily accessible after he begins his practice. This physician is in the most favourable position to help in the control—for he will be best equipped to discover cancer in early stage." (National Advisory Cancer Council of U.S.A.).

Almost all the cases came to us in an advanced stage of the disease when surgical treatments were out of question and radiotherapy also held out very little hope.

Most of these cases came with the following major complaints:

(1) swelling in the neck,
(2) pain and dysphagia,
(3) blood spitting,
(4) hoarseness.

Many of them were for long under treatment, at home or elsewhere, of some form or other, and many of them did not take the condition seriously in the

beginning. Months were spent in gargles, throat-paints, etc. Only recently a case came where the main complaint was pain in the neck and for which 3 valuable months were lost in palliative treatment. On examination here cancer was found in the lateral pharyngeal wall. This case is not included in the above statistics. Therefore, the remarks made by the National Cancer Council of U.S.A. are applicable here also, so far as our general practitioners are concerned.

The symptoms are very trivial in the beginning and so are overlooked. The most unfortunate fact is that these cancers remain silent for a long long time and go on advancing without being detected. The symptoms quoted above are those of advanced stages. One should not wait till the appearance of those signs and symptoms. The general practitioner, if he is conscious of the disease can help in its early diagnosis and thus ameliorate to some extent the terriable sufferings ot the patients.

It would be best to quote the remarks of Thompson and Negus—*in case of the intrinsic carcinoma*—"no one at any age, should be continuously hoarse for more than a few weeks, without obtaining a skilled inspection and an opinion on the larynx."

In case of extrinsic carcinoma "the early symptoms are vague and frequently overlooked. Slight discomfort in the throat or a feeling of something sticking and needing to be cleared by voluntary movement or swallowing may be all that appear."

The important point is that neither the patient nor the practitioner should wait for the appearance of the symptoms later connected with the laryngeal cancer, *e.g.*, dysphagia, salivation, pain in the ear, fœtor, hæmorrhage or cachexia.

Any continuous discomfort in the throat lasting more than three weeks in a patient over 40 years of age (here I should like to add even at 20 years) should call for a thorough examination of the pharynx and the larynx.

It is imperative that every doctor in his undergraduate course should be trained to use the mirrors to look into the pharynx and the larynx. This will enable him to diagnose the disease in its early stage.

Once the diagnosis is made the patients should be advised to make immediate contact with the centres where facilities for treatment exist.

Conclusion

The Bhore Committee already recommended that a cancer research centre should be established in Eastern India. In view of the prevalance of this disease in Assam, the Assam Medical College may form a suitable nucleus for starting such a centre.

Acknowledgment

I have to thank the authorities of the Assam Medical College for kindly permitting me to utilise the figures of the hospital and my House Surgeon for helping me in collecting them.

CASE NOTES

TWO CASES OF TYPHUS FEVER

A. K. GHOSE, M.B.

Guptipara, West Bengal.

I am giving below two case reports of typhus fever occurring simultaneously in the same house at Guptipara, Hooghly, West Bengal.

Case 1—R. C. Bhowmick, aged 25 years, H. M., seen by me on 15-10-50 had fever for 8 days. The temperature ranged between 101°F and 103·5°F. On the 5th day of his illness he developed red maculopapular eruptions over the trunk and limbs specially marked over the arms and forearms. The eruptions were discrete. There was no eruption over the face which appeared swollen and congested. The eyes were congested. There was headache of some severity and pain all over the body. There was cough with slight expectoration. The patient was constipated. Prostration was marked from the beginning of his illness.

On examination, on 15-10-50 at 8 A.M. the patient looked ill and somewhat toxic. The temperature was 101°F, pulse 100 per minute; respiration 20 per minute; breathing quiet. The patient was conscious; pupillary reflexes were normal; there was no rigidity in the neck or limbs; Kernig's sign—absent; tongue—dry and furred; spleen and liver—not palpable and slight tenderness all over the abdomen was present. Heart—nothing particular; lungs—scattered rhonchi over both the lungs.

Blood slides taken on 16-10-50 and examined did not reveal any malaria parasite. In view of the symptoms of continuous fever, headache of some severity, distinct eruptions and early prostration typhus fever was suspected. The blood was taken for Widal and Weil-Felix reactions on 22-10-50 (13th day of illness). The report of the examination was: Widal negative for typhoid, para A and para B.

Weil-Felix reaction OXK positive upto 1/250; OX 19—negative; OX_2 positive upto 1/25. So, the diagnosis was typhus fever (mite borne). The temperature gradually came down by lysis on the 17th day and the progress towards recovery was satisfactory. The treatment was symptomatic and no specific drug was used.

Case 2—The other case Jogmaya Kar, aged 15 years, H.F., occurring in the same house, first seen by me on the same day (15-10-50) as the previous case, was on the 7th day of her illness. She had similar eruptions on the 5th day of her illness and the temperature ranged between 102°F and 104°F. This patient complained of severe headache and she was at times delirious. The prostration was very marked; there was tremor in her hands and tongue. The pulse was weak. In fact, all the symptoms of the previous case were much more marked in this one.

The treatment instituted was symptomatic and supportive; coramine 1·7 c.c. I. M. every twelve hours; rectal saline with glucose by drip method ; Berin 100 mgm. I. M. every day; calcium and vitamin C were also given. The patient took a better turn on the 13th day and was completely afebrile on the 14th day. Thereafter the progress was satisfactory.

No blood examination was done in this case but having marked and similar symptoms as the previous case and the termination of fever and other symptoms being abrupt, it can be said with fair amount of certainty that this case was also one of typhus fever.

AUREOMYCIN IN APPENDICITIS

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On 22-12-50 I was called upon to see a case of pain in the abdomen with irregular motions unsuccessfully treated for about 12 days in a village hospital. The patient was a stout Hindu male aged 36 years.. A cultivator by occupation, he was living mainly on vegetarian diet. He felt pain in the lower abdomen in the midnight of 10-12-50. He had attended a feast that night. About an hour later he passed two loose motions but the pain was not relieved. Local medical advice was taken and he was treated with sulphathiazole tables and a mixture containing tincture hyoscyamus and tincture belladonna on the suspicion of the case being one of dysentery. He was mainly treated for dysentery for about 12 days with different ayurvedic and allopathic drugs without any relief and later was transferred to the city for treatment.

The patient's main complaint related to pain in the lower abdomen with loose motions. On examination the tongue was thickly coated, pulse rate 148 p.m.—volume and tension fair; temperature 100·8°F; no history of vomiting; general condition fair but look anxious. There was definite muscle guard in the right iliac fossa. McBurnev's point was tender and a lump was also felt. A provisional diagnosis of appendicular lump was made and the following treatment was started.

Aureomycin 2 capsules 6-hourly was prescribed. This was the only drug given but night's rest was assured with hypnotics and food was restricted to only fluid drinks. By the third day the patient's condition was definitely better. Pain disappeared. The temperature came down to 99·2°F and pulse rate was 97 p.m.; general look was better. A liberal supply of fruit juice and Horlicks was allowed as the patient became hungry. By the 6th day the lump disappeared and the patient was for all purposes quite fit except that he complained of a little weakness.

The patient was advised to go home and return for operative treatment after six weeks.

ACUTE ABDOMINAL SYMPTOMS WITH PALUDRINE

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Gorakhpur.

Paludrine is one of the most widely used antimalarial drugs. Though paludrine has got many advantages, it is not without toxic effects, one of which that has come under the observation of the author being 'acute abdominal pain' often simulating a case of acute abdomen.

Case Reports

Case 1—Mrs. L. B., a young lady of 17 years, got acute attack of fever which came with rigor and rose up to 103°F; there were headache and pain in the limbs. Blood was examined and B. T. rings were found. She was given one tablet of 100 mgm. of paludrine at about 9 A.M. The dose was repeated at 2 P.M. After fifteen minutes (2-15 P.M.) she complained of pain round about the umbilicus which soon became localised in the right iliac fossa and later on generalised. The abdomen was rigid and the tenderness marked throughout the lower abdomen. She soon became uncontrollable, was tossing on the bed, restless and delirious. She started vomiting and the pain would subside a bit with each vomiting which would again start just after few minutes. I was called upon to see her and I found her intensely ill and I thought of appendicitis. Rectal examination was refused. Total white cell count was only 6,800 c.mm. I kept the patient on conservative line. The head end of the bed was raised; nothing by mouth was given; turpentine stupes on the abdomen, sulpha drug and paludrine also given per mouth. The patient, however, did not take the drugs as she was in much agony. I went to see her after 4 hours and found her asleep and was told she was better from 6 P.M. *i.e.* 4 hours after taking the 2nd dose of paludrine. The temperature was still there. She was afraid of the pain to such an extent that she refused taking any drug by mouth and I had to give quinine by intramuscular injection. She was free from malarial symptoms within a few days of treatment with quinine.

This very patient got another relapse a few months after. I repeated paludrine. The same disaster followed. Thereupon, I became a bit conscious about paludrine with that particular patient and whenever she gets a relapse, she plainly refuses paludrine and I too am afraid of giving her the drug. She is all right for the last 5-6 months with treatment with other antimalarial drugs.

Case 2—Miss R. R. S., a girl aged 19 years, fever with rigor on alternate days and sweating when the temperature comes down, headache, anorexia and loss of weight. As a routine the blood was examined and B.T. rings were found. She was given paludrine. After 3 tables of paludrine she complained of severe colicky pain in the abdomen in the ileocæcal region and

vomiting and the condition remained for full 6 hours. I tried belladenal tablets, etc. to check the pain and this did not help. She was in such an agony that morphine grain ¼ had to be given for relief of the pain.

This patient too behaved exactly like the first when paludrine was repeated. She is now all right and has improved in health with other antimalarial drugs, *e.g.*, quinine, chloroquin, etc.

Case 3—Mr. S. R. B., a young M.Sc. student of 20 years, had fever with rigor. Gorakhpur being a heavily malarious place, I examined the blood; B.T. rings were found in plenty. He was given paludrine at 8 P.M. With a single dose he behaved so badly surpassing the previous two cases and was restless and crying throughout the night. All my attempts to control the pain in the abdomen and vomiting were useless till next morning when he felt a little better. I promised not to use paludrine again but I do not know why I prescribed to this patient prophylactic dose of the drug to be taken once weekly. Fortunately he has never behaved in the same manner as he did with the first dose thus differing from his predecessors.

Case 4—Dr. R. N. C. a young man aged 25 years, suddenly got an attack of fever with rigor, aches in the body and headache. Blood was examined for M.P. It was negative. Influenza mixture was given with no effect. Though the blood was negative for M.P. a therapeutic trial for malaria with paludrine was thought of and it was administered. He too behaved exactly similar to the other cases reported and this time the pain was very severe, continuous and the patient was very restless. I tried belladenal tablets etc. to check the pain but nothing short of morphine grain ¼ gave him relief.

Case 5—Mr. K. C. G. a medical student, aged about 23 years, had fever with rigors on alternate days. I examined the blood and it was also examined by my other colleagues in the Pathology Department. Blood was heavily infected with B.T. rings. He was admitted in the students ward. Next day when we went to him to have some slides for demonstration to the students he refused to give blood saying he was very ill last night and had severe pain, so much so that he had to be given morphia and he was exhausted. On enquiry he said he had paludrine. One thing more with this patient was that peculiarly enough he had hæmaturia and on examination no other abnormality could be detected and cause of all this was attributed to paludrine.

Comment

The idea of this article is not to decry the reputed drug or the treatment of malaria with this drug, but just to know from colleagues in the profession whether they have similar experience with the use of the drug or whether these were mere manifestations of some sort of allergy or idiosyncrasy and if it is not due to the drug how do they explain the symptoms noted above.

POST-GRADUATE TRAINING FOR DOCTORS

INDIAN MEDICAL ASSOCIATION SELECTS PERSONNELL

The Indian Medical Association through the good offices of its General Secretary, Dr. S. C. Sen, who was making contacts with representatives of different countries at the World Medical Association meeting and Commonwealth Medical Conference could secure facilities for young Indian doctors for post-graduate training in different hospitals abroad. After such arrangements were finalised for 1951 applications were invited through Provincial Branches of the Association from those who wanted to avail themselves of these opportunities. A Special Selection Committee interviewed the candidates and selected them. After these names were approved by the Hospital authorities where they will be detailed for work, the candidates were informed. They had to meet their own passage expenses. Costs of boarding and lodging and also pocket expenses are provided by the training institutes.

The following doctors have been selected this year by the Indian Medical Association for post-graduate training abroad. Their respective subjects of study and residencies overseas are indicated below:

Dr. S. C. Kuppanna of Bangalore (*Radiology*), Dr. Subba Rao Kakarla of Visakhapatnam (*Radiology*), Dr. G. P. Sharma of Patna (*Pathology and Bacteriology*), Dr. B. G. Achar of Madura (*General Surgery*), and Dr. K. V. S. Rao of Patna (*General Surgery*) will study at the Bronx Hospital, New York, U.S.A.

Dr. G. V. Talwalkar of Bombay (*Pathology and Bacteriology*), Dr. G. C. Sharma of Jaipur, Rajasthan (*General Surgery*), Dr. T. R. Motwani of Bombay (*General Surgery*), Dr. A. A. Carvalho of Ahmedabad (*Dermatology*), and Dr. M. T. Khilnani of Bombay (*Radiology*), will study at the Mount Sinai Hospital, New York, U.S.A.

Dr. S. Durwaish Mohiuddin of Hyderabad-Dn (*E.N.T.*), Dr. R. D. Kamath of Bombay (*Anæsthesiology*), Dr. G. P. Keshari of Patna (*E.N.T.*) and Dr. M. V. Ramachandra Charyalu of Madras (*E.N.T.*) will study at the Royal Victoria Hospital, Montreal, Canada.

Dr. S. P. Ghosal of Banaras (*Mental Diseases*) will study at the Grangegorman Mental Hospital, Dublin, Ireland.

Dr. K. I. George of Quilon (*Orthopedic Surgery*) will study at the Nassau Hospital, Mineola, New York, through the World Medical Association.

JOURNAL OF THE
INDIAN MEDICAL ASSOCIATION

CALCUTTA, AUGUST, 1951

HERMAPHRODITISM

The word 'hermaphrodite' denotes an individual in whom the sexual gonads characteristic of both the sexes, ovary and testis, are represented. The ovary and the testis may exist as separate structures or may be present as a combined gland (ovotestis). It is indeed extremely rare to find an individual who truly possesses both the male and female characteristics with perfect anatomical and functional gonads of the two sexes. Keller[1] classified them as true hermaphrodites and the imperfect forms as pseudohermaphrodites. Cases of true hermaphroditism are indeed extremely rare and have been reported by only a few authors like Webster, Young, Masson and Sheppard.[2] Young,[3] in 1937, collected only 20 cases of true hermaphrodites from literature. It is however more common to find persons having gonads of one sex internally but with the external characteristics and habits of the opposite sex. In accordance with the classification of Klebs (*loc. cit.*) that is usually followed, persons having testes as gonads are called *pseudohermaphroditismus masculinus*, and persons with ovaries as their gonads are called *pseudohermaphroditismus feminis;* each variety being further subdivided into internus and externus. The main interest, difficulty and complication in these pseudohermaphrodites centre around the fact that due to this condition, there is frequently much uncertainty in the determination of sex of the individual, not only at birth but more specially after puberty when the secondary sex characteristics begin to be differentiated. This obviously leads to great disturbances in the habits, mental make up and the social status of the individual for which much attention has, of late, been focussed to the problem of pseudohermaphrodites leading gradually to a better understanding and management of these cases.

The word "hermaphrodite" is derived from the ancient bisexual Greek god, Hermaphroditos, mention about whom is found in a work of the philosopher Theophrastus[4] (300 B. C.). The idea of androgynous deities are found in the traditions and religious concepts of many ancient races like the ancient Egyptians, Jews and other orientals. Other ancient authors like Hipprocrates,[5] Herodotus[1] and Pliny the Elder[1] also mention about hermaphrodites in their writings. Aristotle[6] (384-322 B. C.) referred to human hermaphrodites in his writings, Galen (129-201 A. D.) knew about them and made embryological researches with male and female "semen" to find out the causation of such creatures. Mention about human hermaphrodites are found in the works of Ambroiss Paré[1] (1510-1590), Albrecht von Haller[1] (1708-1777) and many other workers on natural history, biology, anatomy and medicine throughout the eighteenth and the nineteenth centuries; great advancements having been made since the discovery of the microscope and the demonstration of the living animal cell.

Although hermaphroditism, even of the incomplete type, is comparatively uncommon in man, it is however easily realised that since hermaphroditism represents a less specialised form of sex differentiation, it may be regarded as a more primitive form. Evidence of this is plentiful in the lower vertebrates and many varieties of invertebrate animals, specially of the lower order. The earthworm, the edible snail, fresh-water pond mussel, various insects and butterflies very often exhibit genuine hermaphroditism. Certain well known types of frogs and toads very often change from females suggesting that, in them at least, the ovary represents an intermediate stage of development from the originally neutral gonad and that the testis represents the final stage with complete transformation. Certain varieties of swordfish, certain types of birds, pigs have been known to exhibit natural or spontaneous bisexualism by transformation from one sex to the other with an intermediary intersexual stage in between. Hermaphroditic cows known as "free martins" have been known from a long time and formed subjects of study of such eminent men as John Hunter[7, 8] and Antonio Scarpa.[9]

The sex of an individual is indicated by the gonads but are pre-determined by the chromosomal pattern assumed by the zygote formed after fertilisation of the ovum by a spermatozoon. Hertwig[1] and others have shown long ago that the male sex cell is heterogametic, with the specialised X and Y chromosomes, while the female sex cell is always homogametic. The sex determination of the future embryo is effected through the reduction division of the X and Y chromosomes which consistently carry the factors for the two sexes accordingly as the special chromosomes are of the X or of the Y variety. The sex of an individual is, in this way, genetically predetermined, but during embryonic development the differentiation of the primordial sex cell is effected partly by the environmental tissue consisting of a cortex and a medulla, the former of which determines femaleness and the latter maleness of the fully developed gonad.[10] It is believed that either type of gonad is capable of producing both male and female sex hormone but that normally one variety predominates while the other variety is suppressed. During embryonic development, it is thus always a possibility, although actually a rarity, that the sex cell starts to develop in accordance with its pre-determined genetic pattern, but that after a certain stage of development, a reversal takes place (reversal point, "drehpunkt" of Goldschmidt)[11] and the gonad then continues to develop in the direction of the opposite sex, giving rise to various grades of intersexuality. This is the basis of Goldschmidt's quantitative genetic theory. According to the time or stage of development at which the switch over takes place, different grades of inter-sexuality result, and for these individuals affected by such hermaphroditism Goldschmidt (*loc. cit.*) uses the term *intersex*, of whom he classifies five grades—the beginning, the weak, the

middle-strong the strong and finally the complete sex reversal. Goldschmidt's theory explains the very large incidence of male pseudohermaphrodites representing intersexes of a more successful type, but does not apparently explain the frequent occurrence of pseudohermaphrodites, suggesting complete reversal, as compared to the very rare incidence of cases of true or genuine hermaphrodites indicating incomplete reversal. The female pseudohermaphrodites are perhaps not to be considered as intersexes in the above-mentioned context, and Berner[12] thinks that they possibly represent individuals affected by some sort of a virilising process (due to adrenals or other factors) during fœtal life. In the experimental fields, it has been possible to effect the transformation of sex in various forms of lower life, invertebrates and even in vertebrates like chick embryos. The stimuli used for such experiments have been food, environment, variations of temperature, sex hormones[10] and gonadal transplantations.

The study and management of cases of pseudohermaphroditism pose many problems and are very interesting at the same time. Published reports, specially a recent one by E. C. Hamblen[13] and others of Duke University have helped a great deal towards a better and fuller understanding. The importance of a careful examination of the genitals of the newborn specially females, cannot be overstressed although this is very often neglected. In cases of doubt further studies should be carried on for years together, before the sex is finally settled. In cases of doubt, perhaps it is safer for the baby to be brought up as a male rather than a female; for an imperfect male is less likely to enter into the marital relation than an imperfect female. The case of the athlete Vivian Leigh supports this contention. She won many laurels as an outstanding woman athlete in the 1939 Olympic meet, but had to surrender all her titles having "changed" from a woman to an ordinary male. The determination of the genetic sex of hermaphroditic individuals is not at all easy, and in most of the cases this is done after operation and a biopsy study of the gonads. Hamblen and others (*loc. cit.*) have recently been able to diagnose a case of a post pubescent female pseudohermaphroditic by evidences of essentially normal urinary gonadotropins and very high values of urinary 17-ketosteroids. Hamblen and others have shown that hypergonadotropuria does not develop in male pseudohermaphrodites but that subsequent progressive degeneration of the seminiferous tubules results in impaired release of an inhibiting hormone—an œstrogen secreted by Sertoli cells—which monitors the secretion of the follicle stimulating gonadotropin (F. S. H.) by the pituitary. The hypergonadotropuria occurs despite the hyperplasia of apparently normal looking Leydig cells which are supposed to secrete androgens. These male pseudohermaphroditic patients are often hypoandrogenic and usually have low urinary 17-ketosteroids although some subjects may secrete this in increased amounts. Most male pseudohermaphroditic patients also have gynecomastia probably caused by an abnormal steroid metabolism involving the adrenal cortex.

The management of hermaphroditic patients deserves a serious consideration of the habitus, psychosexual orientation of the patient and with correct appreciation of homosexuality likely to develop, or be accentuated, after treatment. Usually it is wise to maintain, and help to develop, the sex of the patient certified at birth, rather than trying to change things so as to adjust the patient's body and mind to conform to the sex indicated by the gonads. This often necessitates ablation of the gonads—done with requisite permission and adequate explanation—followed up later on by administration of suitable hormones.

The study of the subject of hermaphroditism, although much advanced today, is as yet far from being complete. Many are the problems that have still to be solved, concerning this interesting issue. Further researches, experimentation and pooling of informations will certainly lead to a better and fuller understanding and happy solution of this subject.

[1] KELLER, R.—Quoted by, in CIBA monographs, No. 7, Bombay, May, 1950.

[2] WEBSTER, MASSON, YOUNG & SHEPPARD—Refd. in DAVIS' Gynecology and Obstetrics, Vol. II, Chapter 8, p. 1, W. F. Prior Co., Hagerstown Road, U.S.A.

[3] YOUNG, H. H.—Genital abnormalities, hermaphroditism and related adrenal diseases. Willims & Wilkins C., Baltimore, 1937.

[4] THEOPHRASTUS—in his work "Characters".

[5] HIPPOCRATES—in the sixth book of Epidemics.

[6] ARISTOTLE—On the Generation of Animals.

[7] HUNTER, J.—Account of the Free Martin., Philos. Trans. R. Soc., London, *69*/1, 279-293, 1779.

[8] HUNTER, J.—The works of J. H., with Notes, edited by James F. Palmer, **4**, 34-43, London, 1837.

[9] SCARPA, A.—Observazione anatomica sopra un vitello-vacca detto dagl'inglesi freemartin. *Mem. di Matem. e di Fisica della Soc. Ital.* 2/II, 846-852, 1784.

[10] DANTSCHAKOFF, V.—Das Hormon im Aufbau der Geschlechter, Biol. Zentralbl. 58, 1938.

[11] GOLDSCHMIDT, R.—Die Zygotischen sexuellen Zwischenstufer und die Theorie der Geschelchtsbestimmung. Ergebnisse der Biologic. Vol. 2, Berlin, 1927.

[12] BERNER, O.—Hermaphroditismus und sexuelle Umstimmung. Vol. 5, 1938.

[13] HAMBLEN, E. C. *et al*—*Am. J. Obst. Gynec.* **61**:1-19, Jany., 1951.

FIFTH GENERAL ASSEMBLY OF THE WORLD MEDICAL ASSOCIATION

The World Medical Association will hold its Fifth General Assembly at Stockholm, Sweden, from 14th to 21st September, 1951.

The General Assembly will be preceded by a meeting of the Council to be held between 10th and 14th September, 1951. Visits to hospitals, institutions of social welfare and medical centres will be available for those who desire to see these institutions. On 21st September there will be a meeting of the Medical Editors of the World.

CURRENT TOPICS

FOURTH WORLD HEALTH ASSEMBLY SESSION

With a record participation of delegations from 70 countries and observers from 20 international organisations, The Fourth World Health Assembly concluded its session in May last. This year's President of the Assembly was Dr. Leonard A. Scheele, United States Surgeon General, who succeeded India's Minister of Health, Rajkumari Amrit Kaur.

Outstanding among the many accomplishments of the Fourth World Health Organisation, was the adoption of International Sanitary Regulations which, without the need for ratification by Member Governments, will come into force on October 1, 1952, and will replace all existing sanitary conventions for health protection in international travel and trade.

The new code has been drafted in the light of recent advances in knowledge regarding epidemiology and techniques, and it removes avoidable restrictions on international travel and commerce. The requirements in respect of vaccination certificates have been made simpler and less irksome than those under the existing conventions. A notable advance is the machinery provided for continuous study of the working of the regulations, and for settlement of disputes and questions arising from the operation of the new code.

The next year's (1952) working budget was fixed at almost $7,700,000 as compared with this year's corresponding figure of $6,160,000, thus representing an increase of approximately 25%. In addition, about 22% of the total sum contributed by governments to the UN expanded programme of Technical Assistance for Economic Development will be available to WHO next year. The exact amounts which will be expended by UNICEF for its health programmes, under the technical guidance of WHO, is not yet known but is expected to be in the order of $5,000,000.

The 1952 Programme provides for a bold attack to be made on the world-wide problem of the shortage of skilled public health workers. A relatively large number of educational projects will be carried out, covering the utilisation of various methods for promoting the exchange of knowledge and skills, among them individual fellowships, aid to medical institutions, study-groups, seminars, and conferences.

As the Assembly neared its conclusion, a strong resolution was adopted stressing the acute dangers to the health of millions as a result of the growing shortage of DDT and other insecticides essential for the continuation of disease-control programmes now in progress. In the case of malaria alone, the Assembly learnt, more than fifty million people depend for their continued well-being on an adequate supply of the chlorine-based insecticides. The Assembly urged the governments of major producing countries, including the United States, Great Britain, Canada, France and Switzerland, to take all necessary steps to assure that adequate supplies of these insecticides were maintained and were available for export to the countries needing them.

An outstanding new feature of this year's Assembly was a series of informal technical discussions on the education and training of medical and public health personnel. Held early in the session, these discussions served to emphasise the pressing need for certain changes in the curricula of medical schools and related educational institutions and for a reorientation of teaching in these fields toward greater stress on preventive medicine and the individual's health in relation to his total environment.

With the admission of three countries—Japan, Spain and the German Federal Republic—during the course of the Assembly, the membership list of WHO was increased to a total of 78 countries, including the ten which have suspended active participation in the Organization's work. Once again the hope was expressed that these ten would soon find it possible to resume their collaboration.

Also on the proposal of India, plans for providing additional funds to WHO through voluntary contributions began to take shape with the decision, to devise a WHO seal for worldwide use. These "World Health Seals" will be placed at the disposal of member governments willing to arrange for their sale to the general public on a purely voluntary basis. Proceeds will be divided between WHO and the Government, the latter taking 75% of the money for the use of their own health services.

Switzerland was again selected as the seat for the next World Health Assembly, in 1952, but it was suggested that in the future, meetings of the Assembly should be held away from Headquarters every other year with a view to bringing about closer cooperation with the peoples in the member countries.

DR. L. A. SCHEELA'S ADDRESS

In his address delivered at the final plenary meeting of the Fourth World Health Assembly in Geneva on 25th May, 1951 this year's President, Dr. Leonard A. Scheela said:—For nearly a century we have made efforts to control the spread of communicable disease across national borders by international agreement, cooperation and action. Many of these efforts were fragmentary and they left much to be desired. Too often very few nations participated, but we should be proud of every one of those efforts and organizations because each one represented a milestone in our long-range effort to visualize public health as something transcending national boundaries.

We were youthful and inadequate in our earliest efforts in the field of world health. But by 1945, the nations of the world were mature enough to discuss concepts of new and expanded action in international health. The latest and greatest of man's efforts in the international health field began in San Francisco in the United States of America in that year when United Nations was being founded. There, representatives of

several nations suggested that a special conference be convened to consider the truly international problem of health. During the following year a draft constitution was drawn up for a world health organization. Late in that year the draft was approved by an International Health Conference called by the Economic and Social Council of the United Nations and it became the basis for initial world health action on an expanded scale.

As it emerged from the discussions of the International Health Conference, the WHO Constitution may well be considered by future historians as one of the most daring of all international agreements. It includes several concepts of very unusual character. Above all, it defines health as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity." Thus the traditional frontiers of health were pushed forward and the close relationship between international health problems and economic and social conditions was fully recognised.

Rapidly, then, the World Health Organisation took form. The Interim Commission was formed to carry on until enough nations ratified the Constitution and made WHO a real living entity in the health field. By the summer of 1948 we had an established and operating World Health Organization.

Today the WHO infant has become a full-fledged adult. Each World Health Assembly and each year of life has added to WHO's stature. Our activities are broad. Some of them are almost taken for granted and yet they represent very vital functions. I have in mind such outstanding activities as the standardization of biological products and the work of the Expert Committee which has adopted new international standards for penicillin, heroin, and vitamin E, and formulated recommendations on a wide variety of essential therapeutic, prophylactic and diagnostic agents of animal and plant origin, including diphtheria and tetanus toxoids, cholera vaccines, tuberculin, B.C.G. and streptomycin. The part of WHO in the development of an International Pharmacopœia with uniform rules of nomenclature and dosage for chemical drugs is a major accomplishment. The work of the Expert Committee on Habit Forming Drugs and WHO's work with other international agencies concerned with problems of narcotics should serve as major wedges against the increasing exploitation of large numbers of men, women and children through encouragement of drug habits, a problem currently growing in great proportion in many countries of the world as a result of the increased illicit export of narcotic drugs from several countries. The Sixth Decennial Revision of the International List of Causes of Death will bring us closer to comparative national vital statistics than we have been before. The epidemiologic intelligence service is gradually growing as countries improve their individual reporting programs.

The journals and publications of WHO are assuming new importance. Training programs, seminars, congresses and other meetings on a variety of subjects plus the activities of Expert Committees have been major factors in increasing the exchange of information and scientific knowledge in research and practical applications of research.

Programs to expand knowledge and production of antibiotics and insecticides have made some progress, and stimulation of the use of these products has borne remarkable fruit in communicable disease control.

In the area of organization, many of the early plans and hopes have been realized in the three years since the first World Health Assembly. Most countries are now in Regional Organizations. The Geneva headquarters is excellently staffed in both program and management fields.

The Fourth World Health Assembly was not a meeting characterized largely by stirring speeches and by lost motions. On the contrary, it devoted itself sternly to the task which was put before it and performed its duty with a minimum of flourishes. Everyone knew that this task was to draw up blue-prints for definite and immediate action which will bring greater health and happiness to this embattled earth—plans that through community of effort will bring nations closer together.—*WHO News Letter.*

NOTES AND NEWS

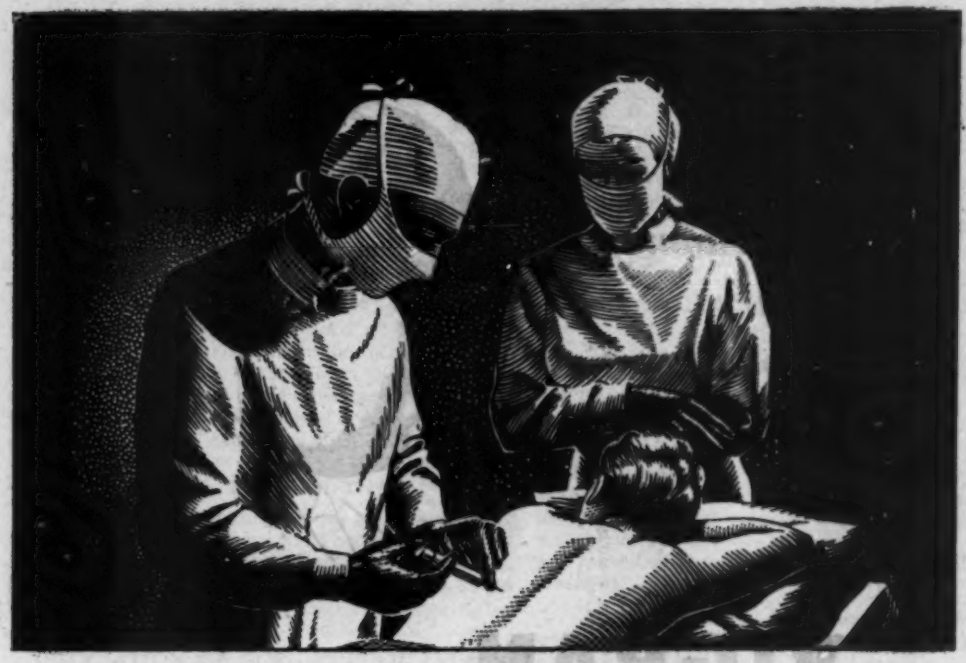
B.C.G. CAMPAIGN FOR RURAL AREAS

Johannes Holm, Director of the International Tuberculosis Campaign, said that the I.T.C. had decided to extend its B.C.G. campaign in India to rural areas also. The campaign is at present confined to cities.

Dr. Holm, who arrived in Madras on 6th March, 1951 from Delhi on his way to Ceylon, said that the new plan aimed at testing and vaccinating, if necessary, every child in India. In East Punjab, where the campaign had already commenced, all the children would be tested in the next three years. Dr. Holm could not state how long they would take to cover the whole country, as it depended on many variable factors.

The work of testing and vaccination had now been completed in Europe, Dr. Holm said.

The efficacy of the B.C.G. vaccination had now been proved beyond any shadow of doubt, Dr. Holm asserted. Reports of observations of children in Finland and Czechoslavakia, two countries where all children had been tested and vaccinated, showed that there was a marked decrease in the incidence of tubercular meningitis, as well as extra-pulmonary tuberculosis—*P.T.I.*



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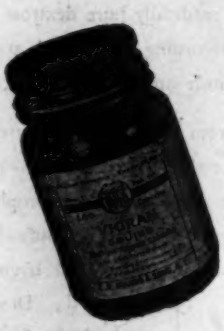

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RAPTAKOS MEDICAL RESEARCH FELLOWSHIPS

The Secretary of the Raptakos Medical Research Board informs that the Board will consider applications for the award of fellowships for research work in recognized institutions on medical and allied subjects. The awards normally consist of Rs. 3,000|- per year for a fellowship and Rs. 750|- per year towards special equipment or chemicals.

Applications should be forwarded through the guides, under whom research work will be carried out, and the Head of the Institution. The applications should be accompanied by six copies of a brief statement of the research project and the comments of the guide regarding the suitability of the project and the facilities existing at the Institution. Fellows appointed should have an M.B.B.S. or M.Sc. degree or its equivalent or not less than 2 years' experience in research work after B.Sc. The awards are made annually and may be renewed on the basis of satisfactory progress.

Applications for grants for the year commencing 1st January, 1952, should reach the Secretary, Raptakos Medical Research Board, C/o Raptakos, Brett & Co., Ltd., Dr. Annie Besant Road, Worli, Bombay 18, before 1st September, 1951.

ASSOCIATION OF SURGEONS OF INDIA

The thirteenth annual session of the Association of Surgeons of India will be held in Calcutta during the last week of December, 1951. Lt. Col. G. D. Kapur, F.R.C.S., I.M.S., will preside over this conference. About two hundred surgeons from different parts of India are expected to attend.

Mr. A. K. Basu, M.S., F.R.C.S., is the local Hony. Secretary from whom all information can be obtained. Mr. L. M. Banerji, M.S., F.R.C.S., is the Chairman of the Reception Committee whose Office is located at the Calcutta Medical Club, 91B, Chittaranjan Avenue, Calcutta.

LUCKNOW UNIVERSITY RESULTS

The Registrar, Lucknow University informs that the following candidates have passed the Diploma in Laryngology and Otology Part I, Examination held in April 1951:—1. Bhupendra Mohan Banerji. 2. Chatar Singh Anand.

GIFT OF TERRAMYCIN TO FIGHT CHOLERA IN WEST BENGAL

Dr. B. C. Dasgupta, Director of Health Services, West Bengal, received on May 23, the gift of Terramycin drugs airshipped from the United States to help fight the cholera epidemic in West Bengal. The actual presentation was made at Dum Dum airport by Sri S. N. Dey, Director, Dey's Medical Stores, Calcutta, on behalf of Charles Pfizer & Company of New York which produced and donated the drugs.

Sm. Vijaylakshmi Pandit, Indian Ambassador in Washington, formally accepted the gift on 6-5-1951 in New York from the original donor.

Dr. B. C. Roy, Chief Minister, West Bengal, had originally requested Dey's Medical Stores, Calcutta, representatives of Charles Pfizer, to buy this antibiotic drug on behalf of the West Bengal Government, but the manufacturers decided to donate a quantity worth Rs. 55,000 to help bring relief to the suffering people of West Bengal.—*U.S.I.S.*

INTERNATIONAL CONGRESS OF PHYSICAL MEDICINE, 1952

The International Congress of Physical Medicine, will be held in London from 14th to 19th July, 1952.

In accordance with the regulations of the International Federation of Physical Medicine, the meetings of the Congress will be reserved for matters dealing with the clinical, remedial, prophylactic and educational aspects of physical medicine and with the diagnostic and therapeutic methods employed in physical medicine and rehabilitation. Exhibitions of interest will also be arranged.

Applications for the programme should be addressed to the Honorary Secretary, International Congress of Physical Medicine (1952), 45 Lincoln's Inn Fields, London, W.C. 2.

NURSING TRAINING IN AUSTRALIA

The problem of adequate nursing training in Australia has been accentuated recently by the shortened training time of nurses due to reduction of working hours, the immediate need for staffing hospitals, the greater number of nurses required as a result of reduced working hours, and by the increased demands on the nurses' time. It may be noted that Australian nurses work a 40-hour five-day week. The establishment of the Melbourne School of Nursing this year, it is believed, will help to provide a solution to this complicated set of problems.

Those taking part in this new educational programme are the Royal Melbourne, Fairfield, Women's and Queen Victoria Memorial Hospital. The Children's Hospital will offer clinical experience.

Lectures, demonstrations and practice will be given in elementary anatomy, hygiene and health, general nursing, history of nursing, ethics and elementary science.

Later lectures and films will cover such subjects as senior general nursing, senior anatomy and physiology, health and hygiene, materia medica and therapeutics, general medical and surgical nursing, operation theatre technique, dietetics, and special branches of medicine and surgery, such as gynæcology, pædiatrics, dermatology, neurology and ethics, including administrative and professional responsibilities.

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors

MODERN TREATMENT OF COMMON OBSTETRIC COMPLICATIONS IN GENERAL PRACTICE

SIR,—In the Special Issue of October, 1950 of your Journal, I read with great interest the article on "Modern treatment of common obstetric complications in general practice" by Dr. G. C. Nandi. I really admire the learned author for his so nicely summing up practically all the important complications with fair details of treatment in the light of recent researches. But I like to respectfully disagree from him on the following points mentioned in the treatment of threatened abortion (page 46).

(*i*) He states, "Morphine and opiates which have been and are still universally used have been found to stimulate rather than inhibit uterine contractions." This is not quite correct. Although there is experimental evidence to suggest that in small doses (*e.g.*, gr. 1/6) morphine may promote increased activity of unstriped muscles,—in more adequate and bigger doses (*e.g.*, gr. 1/3—1/4) it is quite effective in inhibiting uterine contractions and is most important in promoting mental rest which is as important as the physical rest in bed.

(*ii*) As regards the use of Progesterone and Vitamin E, he says that "they are useful but of doubtful value, and if response to treatment is not satisfactory, treatment should not be persevered with. Progesterone is given only when the deficiency of pregnadiol is proved by laboratory tests." Although the use of Vitamin E is harmless but of doubtful value in habitual abortion, its use in the treatment of threatened abortion is, in my opinion, useless in the relative urgency of this condition. The use of progesterone in threatened abortion has been, of late, very controversial and many clinicians (including myself) have found it definitely harmful in many cases in as much as it frequently increases the uterine activity and quickly expels the ovum. The former theory that it inhibits uterine contractions (according to which the writer has also recommended its use to stop uterine contractions in premature labours—page 52) has been strongly questioned and with good reasoning too. It is, however, essential for the maintenance of the decidua for which reason, it may be usefully administered in habitual abortion but not in threatened abortion where bleeding is actually occurring, for it cannot alter the decidua quickly and maintain pregnancy;—on the other hand, if it increases uterine activity, the result will be disastrous. As to the laboratory tests for pregnadiol estimation in urine, I think, the facilities for such estimations in our country are very limited, —and apart from that, it is doubtful whether any safe reliance can be placed on the results of this expensive test.

(*iii*) Lastly, the more controversial therapy of œstrogen in threatened abortion, as laid down by the writer, should have been better omitted,—as the matter is still *sub judice* and no conclusive proof has yet been produced on the efficacy or danger of this recent therapy. I am etc.

Darjeeling,
17-3-51.

SUSHIL KUMAR BHATTACHERJEE,
M.B., B.M.S. (UPPER).

DR. G. C. NANDI'S REJOINDER

DATED 17-4-51

SIR,—I must thank my learned friend for the interest he has taken in my article in the Special October issue of the Journal. He has rightly raised the points of disagreement in the treatment of threatened abortion.

The article being of the nature of a review, I had no scope to express my personal opinion. I merely presented the views expressed in modern textbooks and journals. There is still a great controversy and diversity of opinion regarding the treatment of threatened abortion, especially the role of endocrines.

Regarding the action of morphine, I expressed the opinion of some of the textbooks. J. P. Greenhill in his "Obstetrics in General Practice" (p. 75; 1948) writes: "Morphine is given hypodermically, but it has been shown that this drug stimulates active contraction and does not allay them." Paul Titus in "The Management of Obstetric Difficulties (p. 290; 1945) opines: "It must not be forgotten that morphine has a stimulating effect on smooth muscle fibres, and while relieving the pain of these uterine contractions may actually advance the abortion."

As regards the use of progesterone or estrogen etc., I would mention here the latest view as expressed in the editorial comment in the 1950 Year Book of Obstetrics and Gynecology (p. 63):

"Numerous papers still appear concerning the value of progesterone, estrogens or a combination of these two, thyroid extract, vitamin E and other substances for prevention of threatened abortion. The paper by Colvin and associates is important because it is an analysis of such a large series. The women were given no medication and despite the absence of so-called specific medication the results are practically identical with the glowing reports of individuals who used various hormones, vitamins and other therapy. Only about 4 per cent of their patients could theoretically have been helped by hormone and vitamin therapy.

"Since so many early conceptuses are abnormal, there is no point in continuing hormone, vitamin or other therapy, or bed rest too long. A seven day trial is sufficient. . . . If the patient continues to bleed more than seven days, I see no point in keeping her in bed any longer. She should be permitted to get up and move around, because almost certainly the ovum is abnormal and Nature will expel it any way. When a Guterman pregnanediol test shows low low or decreasing amounts of pregnanediol, I give progesterone, otherwise my only indication for hormone therapy in abortions is habitual abortion, for which I still use diethylstilbesterol, although I am not convinced that it is helpful."

REVIEWS

MEDICAL EXAMINER'S INSURANCE EXAMINATION RECORD REGISTER—Published by Y. R. & Sons, 16/194 Satyanarayanapuram, Buckinghampet P.O., Vijayawada 2, (M.S.M. Ry.), S. India, pp. 185. Price Rs. 5/.

This book contains the usual items, in a tabular form, that a doctor has to fill-in, while conducting the medical examination of a "case" with proposal for Life Insurance. It often happens that, after all the relevant papers are sent over to the Insurance Company with the forms duly filled in, following the medical examination, the doctor concerned has no regular records of his findings and hence may find it difficult to refer to his own examination findings, should it be necessary at any future date. Medical men doing Life Insurance work will find this book very useful in helping them to keep permanent records of their own examination of insurance "cases". Future references to his own records in this book will be helped by the several pages left for "index" to the entries in the subsequent pages of his examination of 185 "cases". Bound in stiff cardboard, the printing, paper and get-up are commendable.

R. S.

ADVICE TO THE EXPECTANT MOTHER ON THE CARE OF HER HEALTH AND THAT OF HER CHILD—By F. J. Browne, Ninth Edition, 1951. Published by E & S Livingstone Ltd., 16 and 17 Teviot Place, Edinburgh. Pp. 50. Price 1s.

This is a reprint of a valuable booklet. It will be of much use to students, obstetricians and their patients.

TRANSACTIONS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA—Edited by Jones R. Gudger, M.D., vol. xxxiii, 1949.

This book gives the proceedings of the 58th Annual Meetings of the Association of Life Insurance Medical Directors of America. This contains in addition to the account of the Transaction of the Association of Life Insurance Medical Director, eight scientific articles.

ESSENTIALS OF ANTIBIOTIC THERAPY—By J. R. Goyal. Published by The Medical Review of Reviews, Burn Bastion Road, Delhi, P.O. Box 1160. 1st Edition 1951, pp. 75. Price Rs. 6/-.

This small volume brings together the present-day knowledge on penicillin, streptomycin, aureomycin, chloromycetin, terramycin, and other antibiotics. The book contains recent informations on the above-mentioned antibiotics and will be useful to the busy practitioners. We hope that the printing mistakes that have crept in this edition will be eliminated in the next edition.

POCKET CAMPANION FOR MEDICAL STUDENTS & PRACTITIONERS—By S. C. Das. Published by Scientific Publishing Co., P.O. Box. No. 969. D/Demy 1/32, pp. 250. Price Rs. 5/8 or 10s.

This little pocket book contains a wealth of information and is divided into eight sections. The informations given are those which one requires by the bedside and should be of much use for medical students and practitioners.

YOUR QUESTIONS

Correspondents should give their names and addresses (not necessarily for publication) and include all relevant details in the questions which should be typed. Questions of medical interest only will be acceptable for this section. They should be sent in a separate envelope marked "Your Questions". Questions of general interest will be published in preference. Lack of space precludes answering all the questions received.

Q. 1. *From where can I get (plus) glutamic acid? Under what proprietary name is it sold? I require it for a mentally deficient case.*

2. *When you are shivering with cold, you take Gur (prepared by heating and drying cane juice); you feel the warmth within five minutes. It shows that this form is also immediately absorbed. Can we not dispense with glucose and thus save our country from the unnecessary drain of money.*

Ans. 1. Glutamic acid in its naturally occurring dextro-rotatory form is available in tablets containing 0·5 gm. (Parke, Davis & Co.). This commercial firm should be contacted for the supply of the material. There is no special proprietary name attached to this material. For recent references to its use in mental deficiency, see article by Zimmerman and Burgemeister (Archives of Neurology and Psychiatry, **65**:291, March, 1951).

2. The feeling of warmth on taking gur may be due to some substance of alcoholic nature (due to fermentation) in gur having peripheral vasodilator action. Intake of glucose and other sugars while one is shivering is not necessarily followed by a feeling of warmth. It is likely that monosaccharides from digestion of gur are absorbed fairly rapidly and may be used with economic advantage in place of glucose but probably oral glucose cannot be dispensed with being a more ethical product.

CURRENT MEDICAL LITERATURE

Some Clinical Aspects of Hepatic and Gall-Bladder Amoebiasis

Ibrahim and others (*J. Roy. Egyptian M. A.*, **62**:538, 1949, Ref. *Trop. Dis. Bull.*, **47**:40, 1950) write that in cases of intestinal parasitization with *Ent. histolytica* the liver is constantly subjected to attacks by amœbæ, most of which it overcomes. If it fails to repell the amœbic invasion one of the following pathological reactions follows:—(1) Simple congestion; (2) multiple amœbic abscesses; (3) solitary abscess; (4) multifocal abscess; or (5) chronic amœbic hepatitis. A very few cases of amœbic cholecystitis have been recorded, chiefly by French workers. Hashim (*Bull. Trop. Dis.*, **31**:379, 1934) stated that he found amœbæ in the mucosa of the gall bladder post-mortem in a case of amœbic liver abscess; Weselmann (*Ibid*, **40**:606, 1943) reported he had recovered vegetative forms and cysts of *Ent. histolytica* from the contents of the duodenum by intubation. It would thus seem that infection of the gall bladder with amœbæ can result from an ascending infection from the gut by the biliary tract.

The authors in a discussion on the symptomatology of amœbic involvement of the liver and of the gall bladder say that acute and subacute cholecystitis of amœbic origin may occur; jaundice is very rare. Some illustrative cases of liver and of gall bladder amœbiasis are described. (The diagnosis in these cases was apparently based on the clinical findings and the response to emetine treatment; in none of them were parasites recorded in the lesions nor was the presence of a gut infection established).

Aureomycin in Intestinal Amoebiasis

Halawani and others (*J. Roy. Egyptian M. A.*, **33**:701, 1950) in a preliminary communication observe:

Seven cases of intestinal amœbiasis were treated with 500 mg. doses of aureomycin every six hours for seven consecutive days, a total dose of fourteen grammes being administered. Five of the cases suffered from parasitic and clinical relapses within a period of forty days after treatment.

Five similar patients received the same dose of aureomycin for ten days, *i.e.*, a total dose of 20 gm. In two cases parasitic relapses occurred within the same period of observation, but no clinical relapses were noted.

The remaining five cases were still asymptomatic and their stools free from *Ent. histolytica* at the end of the period of observation.

No serious toxic effects were encountered in any case.

Synergistic Action of Chaulmoogra and Sulfone Drugs

Tolentino (*J. Philippine M. A.*, **26**:315, 1950) writes:

One hundred fifty-three patients received adequate treatment with chaulmoogra, 40 patients with sulfone, and 33 patients with chaulmoogra-sulfone combination.

Clinically, 37 per cent of the cases among patients considered adequately treated for six months, improved with chaulmoogra, 68 per cent with sulfone, and 42 per cent with chaulmoogra-sulfone combination.

Bacteriologically, 43 per cent of the cases among the same groups of patients, improved with chaulmoogra, 30 per cent with sulfone, and 58 per cent with chaulmoogra-sulfone combination.

It may be concluded, in so far as these results are concerned, that sulfone therapy gives the best clinical but the poorest bacteriological improvements, and that chaulmoogra-sulfone combination gives the best bacteriological improvement, and better clinical improvement than chaulmoogra alone. Hence, chaulmoogra, far from being the obsolete antileprotic medicine that many have already considered it to be, still ranks high in the therapy of leprosy, especially in the elimination of the acid-fast bacilli from the leprotic lesions. In this it is found to be still unsurpassed.

Diseases Simulating Leprosy

Ghosh (*Leprosy in India*, **21**:131, 1949, *Trop. Dis. Bull.*, **47**:48, 1949) writes:

This is a useful clinical description of the diseases liable to be mistaken for leprosy. The most important are the leishmania infections, but oriental sore due to *L. tropica* is found almost entirely in the northernly two-thirds of the western half of India and kala-azar (of which the dermal lesions are relevant) in the remainder of the country. Microscopical examinations of material obtained by puncturing the skin lesions are necessary for their differentiation. Post-kala-azar dermal leishmaniasis is much the more important of the two in this connection because, in addition to the typical dermal nodules, it also occurs in its early stages as small hypopigmented patches on the face, neck and inner sides of the arms and thighs. The patches may coalesce as raised erythematous areas before the more easily recognized nodules form, which are free from lepra bacilli, and they may simulate tuberculoid leprosy. A previous history of a prolonged febrile stage, which yielded to antimony treatment, will be of importance. In dermal leishmaniases, thickened patches most frequently appear around the mouth, and begin with a characteristic flushed appearance.

Tuberculosis of the skin may also resemble tuberculoid leprosy, but in the form of lupus the apple-jelly appearance and the distribution of the lesions enable it to be recognized. The sarcoid form is more difficult to distinguish without biopsy.

Syphilides form a third simulating condition, but the red to copper colour and presence of spots on the palms of the hands and on the soles of the feet are the main distinguishing points. The author states that "blood serum examination" will distinguish the diagnosis and their response to iodides is of value. The presence of thickened nerves and some degree of anæsthesia in tuberculoid lesions of leprosy is also most helpful. Other conditions mentioned as being liable to confusion with leprosy are seborrhœa, acne rosacea and nævus in its various forms.

Gastroenterological Conditions and Complications in the Course of Diabetes Mellitus

Joslin and Risley (*Rev. Gastroenterol.*, **17**:643, 1950) write:

Cancer in the diabetic presents an unrivalled opportunity for early diagnosis and cure.

In the last eleven years, September, 1937 to September, 1948, there were autopsies upon 128 of the authors' cases revealing cancer, and among these were 37 of the patients with cancer of the digestive tract. The distribution was as follows: Mouth and pharynx 4, stomach 7, colon 16, rectum 10. As yet it cannot be proved that the results in the diagnosis and treatment of cancer of the digestive tract are better than for non-diabetics.

The old diagnostic teaching rule that all symptoms should be explained by a single diagnosis falls flat in diabetes. Here one must realise that one is dealing with a group who overwhelmingly are riddled with arteriosclerosis.

The range of age in the 35 cases was from 44 to 76 years of age, the highest incidence being in the 60's. Anæmia was prominent and in many instances the carcinoma was discovered during the course of investigation for anæmia. The average hæmoglobin for the group was 12·4 gm. Twenty-four patients had major surgery in the form of resections. One patient had four separate carcinomas of the colon at different times and has survived four resections.

Ulcerative colitis—nocturnal diarrhœa—Ulcerative colitis is rare in a diabetic clientele. This seems surprising, because diabetics eat a large quantity of coarse food. Nocturnal diarrhœa, however, is not infrequent. Sheridan and Bailey reported 40 cases in 1946. More and more, this has come to be explained on the basis of diabetic neuropathy and the fact that many of these cases improve after months of discomfort confirms it. Undoubtedly the absence of hydrochloric acid and the coarse and large diets are contributory factors, because one seldom, if ever, meets this distressing complication in the diabetically controlled patient. In the above series the injection of crude liver extract was effective in a large percentage of cases. In this connection may be mentioned a recent paper by Rabinowitch, in which the frequency of achlorhydria in diabetes is associated with anæmia and neuritis and a reason therefore presented.

Unobtrusive gastrointestinal emergencies are constantly arising in the cases with diabetic coma. The first to mention is a case with *dilated stomach*—gastric atony. For undiagnosed it can lead to death, just as overdistention of the stomach of an animal will arrest the heart. This is the reason for the standard rule in the authors' clinic in the treatment of coma to be sure to empty the stomach and to do it very, very gently, because the process is a precarious one and carries a hazard. A large stomach tube, is used because the stomach may be retaining coarse food consumed two days earlier. Apparently this condition of gastric atony will result when acidosis—ketonuria—develops, irrespective of whether in diabetic coma or as in childhood following vomiting, as a result of anæsthesia, particularly with the use of ether.

A second gastrointestinal complication of diabetic coma is *gastric hæmorrhage*. Usually this is of old, dark blood, long resident in the stomach. It is most common to find it and so common that it may divert the unwary from the recognition of a peptic ulcer. Unless this blood is removed by lavage, vomiting persists and feeding is useless.

The third complication is a *pseudoperitonitis or a true appendicitis*. This was recognized as long ago as Kussmaul's original description of coma. The symptoms conform closely to peritonitis, this Hippocratic facies, the tense abdomen and even the leucocytosis. The symptoms and signs resemble the condition, more rarely met in the chest, when dehydration in coma is so extreme that a friction rub can be heard. The authors maintain that the depletion of the excretion of sodium chloride in diabetic coma can be demonstrated in part to be due to abnormal functioning of the suprarenal, because that might give a new lead in treatment. Hence the rationale in the liberal use of saline in diabetic coma.

It may be an exaggeration to say that a surgeon is always to see in consultation every case of diabetic coma that is treated in the authors' clinic, but it is not far from the truth, especially when dealing with coma in children. The abdominal distress and distention and the leucocytosis, all common to coma and appendicitis, make the differential diagnosis difficult. The existence of diabetic coma is no excuse for a death from appendicitis, particularly when two hours' observation by surgeon and physician will solve the situation. Both conditions can and do simultaneously exist.

Appendicitis in diabetes is the same as in the non-diabetic, but it is veritably almost symptomless. Some degree of pain and tenderness was present in all cases of appendicitis, and nausea or vomiting in three-fourths. In the acute cases the temperature varied between 98° and 100·2°F, the average being 99·5°F. This finding is similar to the low temperature encountered with diabetics having tuberculosis. The average white count was 16,000 in the acute cases and 20,000 in the ruptured.

Although some patients complained of discomfort rather than pain, there usually was a history of discomfort beginning in the epigastrium which later shifted to the right lower quadrant, thus aiding in making the diagnosis.

Peptic ulcer was almost unknown in the diabetics in the authors' clinic in the early years of the century. This was easily explainable by their short duration of life. Whether it is more or less frequent in diabetics than non-diabetics, it is not known yet. The diabetic diet does not predispose to ulcer. Again, among 10,673 fatal cases there were but 27 instances in which death was attributed to this cause. However, in 332 autopsies upon diabetics of the group between September, 1937 and September, 1948, Dr. Shields Warren and Dr. Philip M. Lecompte encountered 7 ulcers, in two of which the bleeding ulcer led to a fatal issue. Unobtrusiveness of symptoms was characteristic of the group, just as with appendicitis.

Potassium Deficiency in Surgical Patients

Evans (*Ann. Surg.*, **131**:945, 1950, Ref. *Med. Times*, **78**:546, 1950) reports that a study of patients recovering from operation, for whom constant Wangesteen suction with an indwelling gastric tube was used, showed that if such gastric drainage was continued after four or five days, potassium deficiency, as indicated by a fall in plasma potassium below 3·5 MEQ/L, was likely to occur. Potassium deficiency may occur in patients who have been unable to take adequate food by mouth prior to operation, as in œsophageal lesions, pyloric or intestinal obstruction. Fæcal fistula, with loss of large amounts of fluids from the fistula, may also cause potassium deficiency. The possibility of potassium deficiency should be

recognized in surgical cases in which dehydration and salt loss is produced by excessive vomiting, diarrhœa, prolonged gastric suction or other causes; the author has found that if alkalosis (CO_2 content more than 60 volumes per 100) persists in such cases after adequate hydration and administration of sodium chloride, potassium deficiency is to be suspected. If the patient shows extreme muscular weakness and typical electrocardiographic evidence of potassium deficiency (low amplitude of T waves and lengthening of the Q-T interval), rapid replacement of potassium by infusion of KCl solution is indicated; the first few gm. of KCl should be given in one to four hours; after that 2 to 4 gm. daily of KCl above daily losses, given intravenously, is sufficient. Oral administration of potassium chloride is begun as soon as possible; it can be given in the form of tablets, by mouth, or in solution through a gastric or jejunostomy tube. Potassium deficiency in surgical cases can best be prevented by shortening the period of gastric and intestinal drainage as much as possible, and by determining the indications for intravenous fluid therapy by an "intelligent appraisal" of the condition of each individual patient. Illustrative cases are reported.

Sialography in Diagnosis

Rose (*Postgraduate M. J.*, **26**:521, 1950) reviews the history from the first description of sialography by Barsony in 1925 to the papers of Payne in 1931. He has modified the technique which has proved satisfactory in a series of 93 cases.

A blunt number 1 or 2 intravenous needle angled at the upper third of the shaft is used. The Stenson's duct opening can be rendered obvious by massage over the gland. The Wharton's duct opening must be stabilised by fine non-toothed forceps. Neohydriol is used because of its lower viscosity and in rather larger quantities than usual, 1½—2 ml. being injected slowly. For the parotid gland A.P. and lateral are used for the submanidibular.

Clinical Application—The constancy of duct anatomy is emphasised when any distortion becomes significant.

Recurrent pyogenic parotitis: In early cases there is terminal dilatation which may be in one area alone or scattered. The duct itself shows no abnormality and the parenchyma is well outlined. Late cases show typical sialangiectasis, sacculaion of the terminal ducts and beading of the main ducts. The parenchymal shadow is faint or non-existent.

Pneumococcal recurrent parotitis is distinguishable as a type by itself, by marked distortion of the terminal ducts by gross dilatation with no gland outline. The main duct is wider with a "sausage string" appearance. Late cases may reveal abscess formation.

Simple duct obstruction may show a dilatation of the main duct narrowing down to the stricture. There is no involvement of the terminal system and the gland outline is normal.

Duct obstruction with secondary infection which may be difficult to distinguish from long-standing recurrent parotitis, shows the main duct dilated and strictured. The minor ducts share the dilatation but the parenchyma is not involved.

Salivary calculi: The submandibular gland claims all attention. Though sialography may be argued to be unnecessary, still as Feuz (1932) has pointed out 20 per cent of calculi are radiotranslucent and the exact whereabouts of the stone may have to be decided upon.

Salivary tumours: The sialogram is valuable in determining the relationships between glandular and extra-glandular swellings. Intra-glandular swellings may be detected as showing a space occupying lesion unfilled by radiopaque medium, and the main duct may be seen running over the tumour or two adjacent subsidiary ducts may be widely separated. The extrinsic tumours may produce no effects on the gland or a filling defect may be noticed in the profile of the gland.

Problems of Acute Intestinal Obstruction

Wangensteen (*Rev. Gastroenterol.*, **17**:756, 1950) gives in the following lines the summary of his observation on problems of acute intestinal obstruction:

Distention makes the problem of successful management of bowel obstruction very difficult. The chief source of distention is swallowed air. Long sustained intraluminal tension may make a simple obstruction a potentially strangulating one. This occurrence is especially notable in the colon, where, because of the usual competence of ileocecal valve and sphincter, an obstruction in the colon beyond brings about a closed-loop, in consequence of which perforation may occur.

All therapy in obstruction, save decompressive agents, are aimed at improving the patient's condition. The limitation of employment of inlying gastrointestinal tubes to achieve relief of the obstruction without operation, resides in part in the circumstance that the method is not always effective; moreover, it is frequently so slow in achieving satisfactory decompression that, the equanimity of the surgeon and the safety of the patient are threatened thereby; but worst of all, occasionally the method may be inappropriately applied to patients with strangulating obstructions for whom immediate operation is always mandatory.

Nevertheless, suction applied to indwelling tubes has a role and an important one in the management of the distention of obstruction. And when we learn to devise satisfactory technics which will encourage quick descent of inlying tubes to the lower reaches of the distended gut, the usefulness of the method will be pyramided considerably.

If an inert gas with a coefficient of diffusion as great as that of carbon dioxide could be mixed with an adequate amount of oxygen to meet the respiratory needs, the inhalation method of augmenting disappearance of gas from the distended intestine would probably prove useful in the management of many types of intestinal distention.

Operation is still the mainstay of therapy in intestinal obstruction. Great distention is the *bete noir* of the surgeon in dealing with intestinal obstruction. And when surgeons learn to deal satisfactorily at operation with this complication of obstruction, surgeons may some day hope to be able to treat the underlying cause of the obstruction as easily as though distention were not present. At the moment, that goal for many obstructions is beyond our reach; for some it has already been achieved. Continued work and thought upon the matter will help to make that hope and dream a reality. The surgery of acute intestinal obstruction is inching forward.

SUPPLEMENT
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X BENGAL PROVINCIAL MEDICAL CONFERENCE

The tenth Bengal Provincial Medical Conference was held at Baruipur, 24 Parganas, on 18th February, 1951, Dr. A. C. Ukil presiding.

The following are excerpts from the presidential address delivered by Dr. Ukil at the Baruipur Conference:—

I take this opportunity to thank the Reception Committee for having called the Conference for the second time at Baruipur. It is a welcome sign of the times that the members of the profession are taking more interest in the problems of health and welfare of the rural people who constitute 80% of the total population of the province.

The science of Medicine must need recognise the whole nature of man as shaped by his home, his surroundings, his education, his work, his economic status, his recreation and his struggles and aspirations. It has thus become fundamentally a social science and a field of social activity in which is applied practically every basic science directed towards a comprehensive programme of community service. A programme to secure this involves the co-operation and co-ordination of several administrative departments. This has hitherto been neglected in our country. Even the Planning Commission hesitates to sponsor this. My experience in rural reconstruction work has indicated that the people in rural areas are anxious to participate in such a co-operative scheme.

DR. A. C. UKIL, M.B., M.S.P.E. (PARIS), F.S.M.F.B., F.C.C.P. (U.S.A.), F.N.I.
President, X Bengal Provincial Medical Conference, Baruipur.

RURAL HEALTH CENTRES

The Bhore Committee stipulated that the unit at the base should be a health centre for 2 unions, with a population of 20,000. On this basis, there should be 1040 health centres in West Bengal. The West Bengal Government's modified scheme is to have, for the present, a target of 200 centres, of which 75 centres have already been established, including 6 thana hospitals (five 50-bedded and one 20-bedded) out of 250 thanas, and 69 union health centres (25 with 10 beds and 44 with 4 beds each), yielding a total of 696 beds. The wider application of D. D. T. in the prevention of malaria, which constitutes the most important disease in rural areas, may be organised from these centres.

THE RESPONSIBILITIES OF MUNICIPALITY

There are 80 municipalities in West Bengal. Most of them have no planned health schemes and they hardly spend a commensurate part of the revenue in furtherance of health. So far as urban and industrial health is concerned, several contiguous municipalities, particularly in the neighbourhood of the metropolitan area, may combine together in executing the schemes, with the approval of the provincial government. The provincial government should contribute at least 33⅓ to 50% of the expenditure on approved schemes, according to circumstances.

THE BASIS OF MEDICAL RELIEF

333 hospitals in the province provide for a little over 18,000 beds, or 0·7 bed per 1000 of population, as compared to 11 and 8 beds respectively in U.S.A. and England.

The incidence of sickness in this province is much higher than in Western countries, as is borne out by health surveys carried out at Singur and other places. The Singur (rural health) Survey (1944) indicated that 12% of the population were unwell at any time, of whom 1% were acutely ill, 2% chronically ill and over 8% in indifferent health and that 37·4% of the sickness was caused by malaria. Less important causes of sickness, in order of incidence, were measles, diarrrhœas and dysenteries, other fevers, typhoid and paratyphoid, pneumonias, influenza, cholera, asthma, chicken pox,

scabies, tuberculosis, puerperal fevers and venereal diseases. Diarrhœas and dysenteries accounted for 8·8% of all sickness. Nearly 50% harboured hookworm and roundworm ova in their intestines. Some of these diseases can be controlled by the improvement of housing and environmental sanitation and some by protective inoculation. Clinically recognisable malnutrition was found to be widely prevalent, and malnutrition is known to lower the resistance to many diseases and to impair physical efficiency. The rectification of this requires total rural planning involving the co-ordination of several departments of the Government. The sickness rate among school children was found to be as high as 44%. The average duration of disabling illness was found to be 50 days per sick person or 19·6 days per head of the population. It was also found that the public institutions for medical relief in the area provided rudimentary outdoor treatment for only 1/36th of the total sick population. There was only one practitioner of scientific medicine per 5000 of population, in contrast to 4 quacks for the same population. The report concluded that "economic advancement must be solved before material progress in matters of health could be made."

An earlier survey done in Bhopal indicated that 16% of the sick people needed to be hospitalised and that only 4% needed specialised treatment in highly equipped hospitals with specially trained personnel.

Institutional Planning

In this connection, you will pardon me if I quote what I stated 10 years ago on this subject in my presidential address at the Indian Science Congress : "These findings may not apply to all areas and,therefore, each area needs to be surveyed. When this is done it will be found that costly institutions with highly specialised staff will not be needed in such large numbers as now, if the distribution of hospitals and dispensaries is planned in such a way that minor ailments are cared for at the peripheral dispensaries, ordinary hospitals take care of the intermediate categories of cases and the difficult cases are taken to the highly specialised base hospitals. What happens now is that the city hospitals, like those in Calcutta, which possess highly efficient and experienced staff and equipment, deal with all sorts of cases, involving not only wastage of time of the specially trained staff but of money as well.

"To reach medical aid speedily to those who need it and to transport them to larger institutions where the necessary equipment and skill are available, a suitable planning in the development of communications is absolutely essential. It is a matter of regret that communications have not been developed in India to serve the needs of the sick population.

Salary Scales

If the efficiency of the workers in different medical institutions is to be maintained at a high level, they should be given sufficient remuneration for their maintenance and social security and for the education of their children. The rural medical officers of health, who are in charge of the union health centres and who are not allowed to practise, get a consolidated salary of Rs. 150/- P.M., with dearness allowance but with no compensation in lieu of stoppage of practice. Although most of them are licentiates, I consider the scale of salary as grossly inadequate, as food alone costs Rs. 50/- per head per month and as many of them have to educate their children in institutions outside their area. The scale of salaries for officers at the Thana Health Centres, who are mostly graduates, is Rs. 200/- P.M. with a non-practising allowance of Rs. 60/- P.M. These posts should not only have better scales of salary but should be graded and made pensionable, like the B. M. S.

The scales of salary offered to the Bengal Medical Service Officers, upper and lower, should be brought in line with those of other provincial services, such as the Bengal Executive Service (Deputy and Sub-Deputy Magistrates). The Compounders, who perform a very responsible duty, draw a salary in the scale of Rs. 55-1-58-2-100 P.M., which cannot but lead to sub-human conditions of living. It is a serious blot on the administration. The scale of salaries for graduates with D. P. H. qualification should be higher than that of the B. M. S. and adequate non-practising allowance should be attached to the non-practising posts. This anomalous position dissuades the best students from taking post-graduate public health qualifications. It should be removed at an early date.

The Tuberculosis Problem

Our country is now paying the penalty of unplanned industrial phase in development with the result that the incidence of tuberculosis in urban and industrial areas in our province is higher than in many English towns of the same size and population, and it is infiltrating steadily into rural areas. The influx of several millions of refugees of various epidemiological groups into the urban areas have further complicated the position.

In place of 50 urban and 250 rural Tuberculosis Clinics needed in West Bengal, we have not more than a dozen, and where we need at least 50,000 beds for the isolation of infective cases we have barely 1500 beds, almost half of which are provided in Government Institutions, but unfortunately very few of the institutions are fully staffed and equipped for all the types of treatment which are being followed with success in Western countries. There are no Preventoria, no institutions for the treatment of osteo-articular tuberculosis and no arrangements yet for the rehabilitation of partially cured cases. The development of a Thoracic Surgery Unit and arrangements for the training of surgeons, anaesthetists and ancillary staff are urgently needed. The laboratories need levelling up. The institutions should also be provided with arrangements for dental and other types of care needed in them. The B. C. G. scheme needs expansion.

Medical Education

Medical education is directed towards two objectives —educational and vocational. In the former, the student is taught method so that he can collect, analyse and interpret facts for himself and observe and understand what he observes. In the latter, he is given such knowledge adapted to the requirements of the country as will enable him to ably practise medicine on his own account. Contact with active research programmes, or better participation in such programmes, are factors of major influence in the educational development of the student. Unfortunately in the medical colleges in our country research programmes are sadly neglected, partly because of lack of initiative from and provision of funds by the administrative authorities and partly because most of the

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medical colleges have hitherto been outside the sphere of University development, control and co-ordination. The Medical Faculties should be properly constituted and the Medical Colleges should be brought under the financial and administrative control of the University. The whole of the medical and public health education budget of the Government should be handed over to the University, with such supplementary grants, made from time to time, as would raise the status of education in the different medical colleges to more or less the same level of efficiency. The selection and appointment of the proper type of personnel should be left to the University, working through its Faculty. Most Universities have hitherto confined themselves to prescribe the medical curriculum and to conduct examinations on a syllabus.

Most medical colleges are not properly equipped with the tools of scientific progress and the standard of teachers is low, partly because they are poorly paid and partly because they are not properly trained owing to lack of training facilities in the country. Residential arrangements during internship training are lacking. Most medical colleges are deficient in properly functionating libraries, due partly to lack of outlook and partly to inadequate funds being set apart for books and periodicals.

It has been recognised that students receive satisfactory guidance and supervision only through competent teacher : student relationship. The ratio is often 1 : 2 in U.S.A. and 1 : 8 in U.K. The Indian Medical Council advises a ratio of 1 : 10, but in most of the medical colleges in West Bengal the ratio varies between 1 : 36 in Anatomy to 1 : 70 in Hygiene and Public Health and in Forensic and State Medicine. If an all-round improvement of the quality and content of teaching is desired, competent teachers must be appointed in larger numbers and they must be given adequate remuneration. Hitherto it has been the practice in our country to run medical education on the cheap. The sooner it is abandoned the better, as otherwise the quality of service to be given to the people will suffer.

The true objective of medical education demands that all the subjects should be dovetailed into each other so that the student at the end of the course may have received a co-ordinated and intelligent view of medicine and be competent to undertake practice, at first under supervision but later on his own responsibility.

In the hospital wards, the student should take an active and not a passive part. There are far too many students in the ward to make this possible. In-patient clerkships should be so organised as to encourage the student's sense of responsibility for his patients. He should be assigned an adequate variety of cases. The main purposes of out-patient teaching should be to enable the student to observe how an experienced physician or surgeon approaches and treats a patient who is a stranger to him, to provide for the student's exercise in his clinical skill and to demonstrate the importance of "follow up" of patients with the help of a Medical Social Worker. Such an arrangement does not exist in our institutions to-day.

It is evident that a revision of the curriculum and teaching methods is urgently called for. Such revision should ensure that each aspect of medicine receives its appropriate emphasis in a co-ordinated scheme. Each speciality should be taught to the extent and to the standard appropriate to a basic training in medicine. Social Medicine and Psychiatry should, however, be regarded as an integral part of all stages of the training. The present curriculum in Public Health and Hygiene is out of date. A new curriculum should be integrated into Preventive and Social Medicine, which should be an important department of all medical colleges. An urban and rural Health Unit should be attached to all medical colleges for training in community physiology, pathology, diagnosis and prevention with a sociological approach. When this is done, the students will not only be better doctors but will learn better to identify their interests with those of he people.

Post-Graduate Training

Post-graduate training should also form an important component of a medical teaching institution.

The standard, supervision, direction and assessment of the progress of research can be assured by establishing a *Research Board* in each college, under the guidance of the Council of Post-Graduate Teaching in Medical Science. Such a Council was formed by the Calcutta University as far back as the 4th May, 1948, but, alas, no steps have yet been taken to implement the scheme. The inter-University co-ordination of research can be undertaken by the Indian Council of Medical Research, which should form a section of the National Research Council.

A steady supply of medical workers is an important consideration in planning. At the present moment, 455 students are admitted into 4 Medical Colleges, excluding 220 Licentiates for Condensed M.B. Course in 3 Medical Colleges. After the abolition of the Lake Medical College in 1952, the source of training for 100 licentiates in this province will cease. In view of this and as a logical corollary to the abolition of medical schools in this province, their continued training should be distributed over the four Medical Colleges in such numbers and in such manner as not to interfere with the efficient training of the regular undergraduate students. The double-shift scheme introduced some years ago may now be stopped.

Approximately one-third of the undergraduate medical students come out successful at the Final examination. Where two-thirds of students fail, entailing great family and national loss of time and money, there must be something wrong either in the selection of candidates for admission or in the methods of teaching or in the procedure of examination. The matter is too serious to be allowed to rest where it is. A thorough investgiation should be carried out in the matter, with a view to rectify defects, if any.

No medical colleges here possess a *Preliminary Training School for Nurses.* If the services of nurses are considered to be important in the care and follow-up of patients, it is necessary to improve their training. Arrangements should also be made for a Degree Course in Nursing. Their emoluments and service conditions also need careful consideration.

In view of the Government's *policy of utilising the national language* for transmitting higher scientific training within 15 years, it is necessary to take the opinion of the Indian Medical Association on the question. I feel that the Association should appoint a Committee to go into it, with regard to the evolution of scientific terminology and literature. It will not serve the purpose to translate foreign books but to produce books based on data already available in the country.

The Supply of Drugs and Instruments

The cost of drugs in our country is so high that the bulk of the population, who belong to low economic groups, cannot afford to have the scientific treatment. The only way in which drugs can be cheaply produced and brought within the means of the masses is to utilize the local resources and use indigenous products, instead of expensive imported preparations. In spite of the fact that nearly ¾ths of the medicinal plants mentioned in the British and other pharmacopœias grow in India in a state of nature and that others can be easily grown, no attempts have yet been made to systematically cultivate and study Indian medicinal plants, except a small fraction (10%) by Chopra and his school of workers. India has still to depend on foreign countries for the supply of many basic chemicals, needed for the manufacture of drugs, particularly alkaloids, organic and inorganic acids, coal and wood distillation products and various solvents used in the manufacture of synthetic drugs. Government cannot remain apathetic to the serious situation with regard to the economic production of medicaments needed for the present-day therapy. I feel that the problem of making India self-supporting in the matter of her drug requirements is connected with so many interrelated factors that the Government should appoint a committee to examine the position and formulate and execute a programme, including researches.

The medical profession and the people desire that the quality of the drugs produced in the country should be properly ensured. It is a matter of great regret that provincial governments have not yet found it possible to organise their Drug Control laboratories, with the result that while foreign drugs are pouring into our country, owing to defects in the import control policy, our own products are not able to consolidate their reputation and compete with foreign products. The Drugs Act of 1940 is not able to function properly without being supported by these laboratories. Four provinces have thus far started these laboratories but almost all of them are ill-equipped and inadequately staffed.

As antibiotics and sulpha drugs constitute a large bulk of our imports to-day, it is necessary to encourage their manufacture where *pilot-plant studies under Indian conditions* have been successful in order to save a huge drain on our finances by embarking straight on large scale manufacture by importing large plants from foreign countries.

The compilation of a comprehensive Indian Pharmacopœia will go a great way in helping the situation. Although the Government of India has been at it for over 5 years, it is a matter of regret that further delay is occurring in the matter.

The remarks which apply to drug supplies, also apply to the *supply of instruments and x-ray films*. It is a matter of shame that, inspite of most raw materials and competent technical personnel being available in our country, the equipment of hospitals, laboratories and pharmaceutical concerns should have to wait for months and years to get such equipment imported from abroad. Soviet Russia does not depend on any country for her supplies. Why should we?

Amendment of the Bengal Medical Act, 1914

The whole of the Bengal Medcial Act, 1914 should be replaced by modern democratic procedures existing in other countries and similar acts recently modified in India to suit her present requirements. The functions of the Medical Council should be widened to include not only registration and the maintenance of the ethical standards of the profession but also the maintenance of a proper standard of medical education. Proper representation should be given to the various interests involved, including the Indian Medical Association. All foreign qualifications recognised should be on a reciprocal basis.

Wanted a Provincial Health Advisory Board and a Public Health Act.

The Central Advisory Board of Health in India was formed as early as 1937. Before the war, undivided Bengal had a body called the Sanitary Board which was later modified to form the Provincial Advisory Board of Health with, among others, representatives of the Indian Medical Association. As a matter of fact, in 1943, the Bengal Branch of the I. M. A. nominated a member on this Board. Its activities were suspended towards the latter part of the war. The Bhore Committee also recommended the formation of such a Board, and three years ago the Bengal Provincial Medical Conference urged on the Government to form one in West Bengal, but it is a matter of regret that the Government has not yet moved in the matter. The voluntary co-operation of various agencies on this Board will not cost any money to the Government, and yet inexplicable delay occurs.

Madras passed a Public Health Act in 1939. Quick and co-ordinated action cannot often be taken by the administrative authorities with old legislation, which possesses many lacunae. A comprehensive Public Health Act is likely to further many health schemes and will educate the public and secure the co-operation of the medical profession.

Matters Concerning Ourselves

The Indian Medical Association is a craft association of scientific workers, who are connected with all social groups of the country. As such, it is as much interested in medical and public health planning, in relief of human suffering in times of national or artificially created catastrophe, in helping fellow-brethren in distress and in maintaining a high standard of ethical conduct, as in safeguarding the honour, prestige and reputation of the profession. During the last 17 years of its existence, the Association has done a tremendous amount of work in many directions but still much remains to be done.

It is estimated that there are approximately 11,000 qualified members of the medical profession in West Bengal, although most of them are distributed over the larger towns. Yet the membership of the Branch is less than 3000. The volume of membership in an Association such as ours gives the necessary strength to render effective service and to be heard in the councils of the people and the Government. We ought to realise that our strength lies in our unity. I would, therefore, appeal to all qualified members of the profession to enrol themselves as members of the Association.

* * * * *

The following are the resolutions passed at the tenth Bengal Provincial Medical Conference held at Baruipur (24-Parganas) in West Bengal, on 18th February, 1951:—

1. *Food Shortage in West Bengal*:

In the interest of adequate nutrition of the population of West Bengal, this Conference views with alarm, the failure

of the Government to achieve self-sufficiency in food during the three and half years of independence and feels that production of food grains has to be actively encouraged by all possible means including the changing of the existing procurement policy by offering fair prices to the cultivator and taking recourse to modern and improved methods of cultivation and introduction of suitable agrarian reforms.

2. *Rural Health Scheme* :

This Conference recommends to the Government of West Bengal to undertake total rural planning envisaging re-construction in all its aspects including establishment of rural health centres so that the primary necessities of maintaining and improving the health conditions of the rural population constituting 80% of the people of West Bengal may be fulfilled. The Conference is of opinion that planned development of rural communications should receive the highest priority in any such plan so that medical relief may reach speedily the outlying areas and quick transportation of patients to better equipped hospitals from peripheral smaller units may be facilitated.

This Conference is further of opinion that until such time as rural health centres with whole-time medical officers are opened qualified medical practitioners in rural areas should be subsidised and facilities for education of their children be provided by the State.

3. *Insufficiency of Hospital Beds in Calcutta* :

In view of the gross insufficiency of hospital beds in Calcutta, this Conference recommends to the Government of West Bengal to take necessary steps to adequately increase the number of beds in hospitals in and around the city and suburbs of Calcutta and arrange for up-to-date treatment in such hospitals where this is not available and to subsidise the non-official hospitals with sufficient financial aid for the purpose.

This Conference further recommends to the authorities of the Nilratan Sarkar Medical College and Calcutta National Medical Institute of Calcutta to arrange for starting all their specialist departments at an early date during the current year.

4. *Lake Medical College and Hospital* :

This Conference views with grave concern the recent decision of the Government of India to close down the Lake Medical College and its attached 700-bedded hospital, mainly on financial grounds. The medical school standard of education having been abolished, this Conference feels that arrangements on a large scale, for the condensed M.B.B.S. course have to be made for the training of the existing medical licentiates, a large number of whom are anxious to undertake the course. This would not only enable the medical licentiates to obtain better medical training but would also, in the long run, benefit the common people in rural as well as urban areas whom these licentiate doctors are serving.

Experience having shown that the existing system of training the "under-graduate" and the "condensed" medical course students in the same medical college is detrimental to the interests of both these types of students, a separate medical college like the Lake Medical College should be run in Calcutta, on a long-term basis, in order to afford facilities to the civilian medical licentiates for obtaining the requisite training for condensed M.B.B.S. course. The continuance of the Lake Medical College and Hospital will also serve the purpose of provision of a modern large hospital in South Calcutta which is so essential, particularly in view of the fact that the over-populated cosmopolitan city of Calcutta, which is inhabited by several lacs of refugees from Pakistan, is expanding mainly towards the South.

If the existing temporary structures built during the War for the American Army Hospital are not expected to last longer than 1952, arrangements shouuld be made by the authorities concerned to take over the lands of the Calcutta Improvement Trust over which, part of the Lake Medical College is situated, so that permanent buildings may be erected at this site, for lodging the Lake Medical College and its Hospitals.

This Conference further resolves that the Bengal Provincial Council be authorised to take up necessary campaign in co-operation with other interested orgahisation to give effect to the above resolution.

5. *Medical Education* :

This Conference is of the opinion that

(a) Medical Education in this province should be fully controlled by the University of Calcutta through its medical faculty which should be suitably reconstituted and the Government should place adequate funds for this purpose to the University which should see that all medical teaching institutions attain the standard of efficiency in all respects in conformity with the recommendations of the "Medical Council of India".

(b) Adequate provisions should be made for Post-Graduate Medical Education so as to ensure availability of medical teachers for the expanding needs of the province and thus prevent drainage of this country's resources by prospective teachers seeking foreign degrees and diplomas.

(c) Double shift system and training for condensed M.B.B.S. course in the institutions meant for training of under-graduates is not only undesirable from educations viewpoint but is also annoying to the patients in the hospitals where such training is being given.

(d) Arrangements for graduate's course in Dentistry should be made immediately.

(e) In view of the long course of study and training and there being no special post-graduate degree course in the non-clinical medical subjects the M.B.B.S. degree should be treated at par with the master's degree in science or arts for the purpose of higher doctorate degree like Ph.D., D. Phil. etc.

6. *Bengal Medical Act* :

This Conference urges the Government for amendment of Bengal Medical Act 1914 on democratic principles to have majority of elected members to compose it with an elected President.

7. *Unqualified Medical Practitioners* :

This Conference learns with concern that attempts are being made by the unrecognised and unqualified medical practitioners to get themselves not only recognised by the Government but to get registered as full-fledged registered medical practitioners at par with others. In the interest of the well being of the people of this province, the Conference urges upon the Government, West Bengal Council of Medical Registration and Public Leaders not to countenance these

proposals as those if acceded to will mislead and dupe our people.

8. *Manufacture of Drugs* :

This Conference urges upon the manufacturing concerns of this country to pool their resources and evolve schemes for manufacturing essential drugs and allied products to make our country self-sufficient in quality products.

9. *Medical Advisory Committee* :

This Conference feels the necessity of having Consultative Committee on (a) Medical Education, (b) Post-graduate Medical studies, (c) Public Health, (d) Drug manufacture and control, (e) Distribution of essential imported drugs in the interest of the profession and their relations with the public and urges the constitution of those committees by the Government and to have representatives of the Indian Medical Association, Bengal Provincial Branch, on these bodies.

10. *Hospital Planning and Standardisation Committee* :

This Conference strongly feels that time is ripe to set up a Hospital Standardisation Committee under the auspices of the Bengal Provincial Branch of the Indian Medical Association to investigate into the conditions of the Hospitals under Government and corporate bodies with a view to recommend their re-organisation and improvement in the light of the present status and requirements of the country. In order that proper data may be obtained to make the enquiry thorough and sound, this conference recommends that opinion of hospital authorities as also of the public be invited by the Hospital Standardisation Committee.

11. *Health Schemes & Rationalisation of Production of Doctors* :

This Conference views with concern the present anomalous situation in which the common people of the province are suffering acutely for want of proper medical care and assistance, while the majority of the members of the medical profession and particularly the junior members of the profession are finding it increasingly difficult to make both ends meet. The Conference therefore recommends that the Government of West Bengal and the Bengal Provincial Branch of the Indian Medical Association should devise ways and means to rationalise the further production of medical personnel with reference to the health schemes so as to ensure full employment for doctors and adequate medical relief for the population.

12. *Control and Treatment of Leprosy*:

This Conference reiterates the following resolution passed at the 9th Bengal Provincial Medical Conference held in December 1949:—

"This Conference learns with great satisfaction the establishment of a Leprosy Hospital at Bankura by the Government of West Bengal with 100 beds at present and with a provision for gradually enlarging the number of beds to 500.

This Conference, however, views, with grave concern the decision of the Government of West Bengal to curtail the number of beds in the Albert Victor Leper Hospital at Gobra. There was a provision of 200 beds in this hospital for 30,000 Leprosy cases in and around Calcutta of which approximately 25% are infectious. As there is no provision for treatment of people afflicted with leprosy and suffering from other ailments in any other hospital in the city even a provision of 200 beds is too inadequate. This Conference therefore urges upon the Government of West Bengal—

(*i*) to restore immediately the number of original beds in the Albert Victor Leper Hospital with adequate financial grant;

(*ii*) to make adequate arrangements in the said hospital for surgical, medical, gynæcological and other specialist treatment necessary for the various ailments of people afflicted with leprosy;

(*iii*) to start a separate leper home for incurable leprosy cases in the neighbourhood of the city of Calcutta."

13. *Scale of Pay of Medical Officers* :

This Conference is of considered opinion that the recently announced scales of pay for medical men in Government Services fall far short of recommendations of the Indian Medical Association. The discriminatory scale of pay for medical services in relation to other services of the Government *viz.*—administrative, engineering, educational and judicial etc. is entirely unjustified in view of the long hours of duty and constant attendance required of medical men ; and in view of their long years of expensive study.

This Conference therefore recommends that the scale of pay for medical and health services should be revised immediately and should be brought at par with the aforesaid services.

14. *Terms & Conditions of Service of Tea Garden Medical Officers*:

In view of the fact that the salaries and allowances paid to medical personnel of all categories employed at the Tea Gardens under the India Tea Association and the Indian Tea Planters Association are too low compared to those offered in other industries and that conditions of leave and social security are very unsatisfactory, resolved that the Associations concerned be requested to revise the scales of pay and conditions of leave and social security of medical officers serving under them in the light of a memorandum submitted to them by the Bengal Provincial Branch of the Indian Medical Association.

XXVIII ALL-INDIA MEDICAL CONFERENCE

THE WORKING COMMITTEE OF THE INDIAN MEDICAL ASSOCIATION, AT ITS LAST MEETING HELD AT HYDERABAD ON 7TH AND 8TH JULY, 1951, DECIDED THAT THE NEXT ALL- INDIA MEDICAL CONFERENCE WILL BE HELD UNDER THE AUSPICES OF THE ASSOCIATION IN *FEBRUARY, 1952* INSTEAD OF IN DECEMBER, 1951, AT *AHMEDABAD.*

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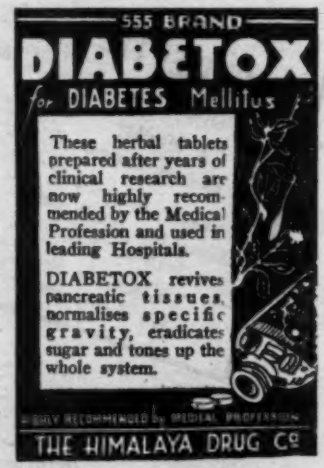
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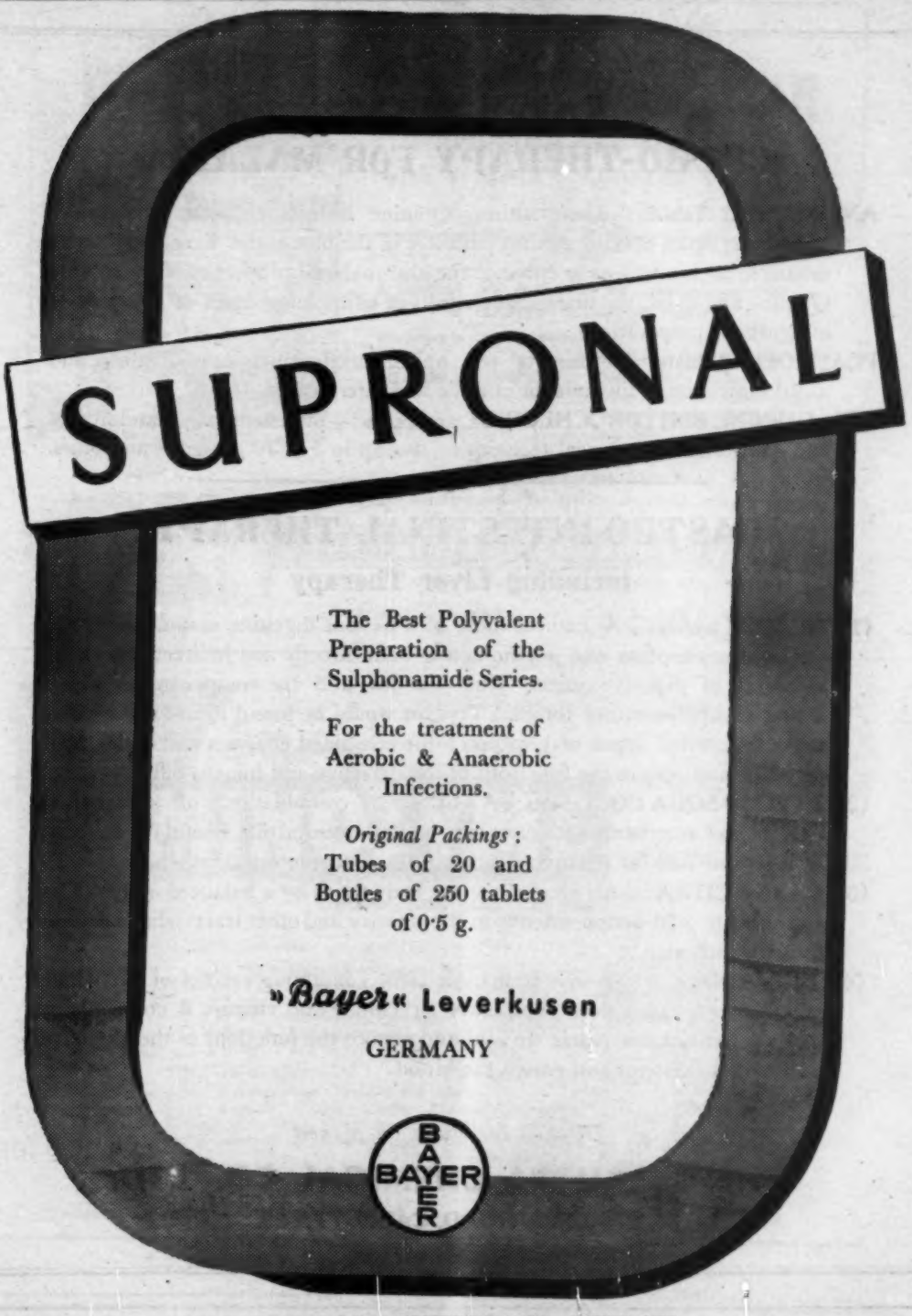

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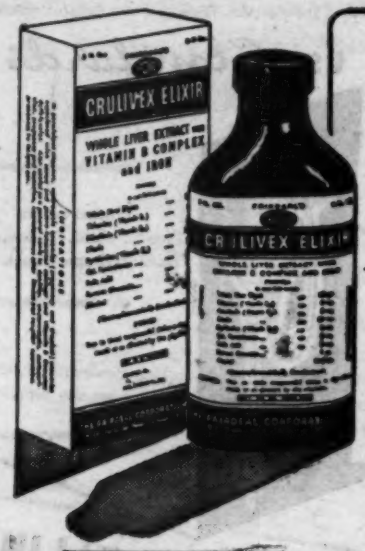

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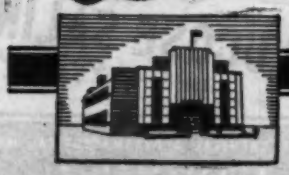

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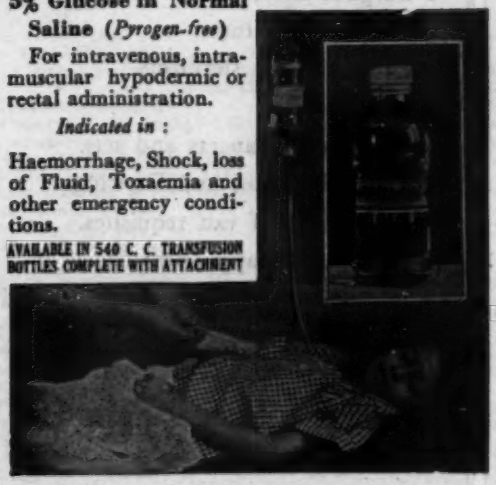

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